THE HOARDING BEHAVIOR AND FOOD INTAKE OF THE HAMSTER FOLLOWING HYPOTHALAMIC AND LIMBIC FOREBRAIN LESIONS

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INTRODUCTION

The general purpose of this study is to examine the effects of limbic and hypothalamic lesions on the food intake and hoarding behavior of the hamster.

In 1954, Stellar presented a neural model of motivation. The model relied on the concept of excitatory and inhibitory hypothalamic center with most basic drive states of the animal controlled by the reciprocal influences of the respective centers. Although Stellar's model did consider cortical and thalamic influences on the hypothalamic centers, no mention was made of limbic forebrain influences. Over succeeding years, however, greater attention was paid to the role of the limbic system with respect to basic drives. Morgane (1964) (ten years after the appearance of Stellar's model) stressed the importance of the participation of the entire nervous system in basic drive behaviors. In particular he emphasized the importance of limbic-hypothalamic-midbrain interactions with respect to basic behaviors such as feeding and drinking.

This view was well substantiated by the work of such researchers as Robinson (1964), Grossman (1964), Fisher and Coury (1964), and Morgane and Kosman (1960). These authors, using both electrical and chemical stimulation, and ablation of limbic forebrain, demonstrated changes in the eating and drinking behaviors of experimental

animals. Nauta (1963) presented an anatomical analysis of limbic-hypothalamic structures which Morgane (1964) developed into a limbic-hypothalamic-midbrain circuit to subserve such basic behavior patterns.

Little work appears to have been done on role of hypothalamic-limbic interactions in more complicated behavior patterns such as the hoarding of food seen in rodents (Smith and Ross, 1953a).

Hoarding Behavior in Rodents

Effects of food deprivation

Wolfe (1939) appears to be the first researcher to study hoarding under laboratory conditions and his design became the prototype for later experiments. It consisted of allowing the animal to enter a runway from its home cage, collect pellets placed at the end of the runway, and return to the home cage. Most researchers have placed some time limit on the amount of time the animal is allowed to hoard. A period of thirty minutes seems to be favored.

Wolfe reported two major findings. Rats raised on what Wolfe claimed to be a nutritionally inadequate diet proved to be inferior to controls when both were tested on hoarding as adults. Wolfe also found that rats raised on a liquid mash diet showed a reduction on adult hoarding.

Further research was carried out on the hoarding behavior of the rat by Morgan, Stellar and Johnson (1943) and Stellar and Morgan (1945). They found that it was necessary to deprive the

animals prior to testing in order to produce a significant amount of the behavior. Indeed, Stellar and Morgan (1945) concluded that deprivation was the most important factor in initiating hoarding. Most researchers using the rat have tended to use pre-test deprivation to induce the behavior. Deprivation does not seem to play a very important role in the hoarding behavior of the mouse or hamster. In fact Smith and Ross (1953a) report that deprivation reduces the amount of pellets hoarded by the mouse. A period of deprivation does appear to increase hoarding in the hamster (Smith and Ross, 1950, Bunnel and Matalka, unpublished), but deprivation is unnecessary to initiate or maintain a high-level of hoarding in the hamster (Waddell, 1951, Bunnell and Matalka, unpublished).

Deprivation as weanlings appears to influence adult hoarding in the rat. Rats placed on a food deprivation schedule after weaning hoarded a greater number of pellets than non-deprived controls when both groups were tested as adults (Hunt, 1941, Hunt, Schlosberg, Solomon, and Stellar, 1947). On the other hand, McKelvey and Marx (1951) using both male and female rats reported that food and water deprivation of weanlings did not appear to increase their hoarding when tested as adults. However, there did appear to be a clear increase in the hoarding of the weanling food-deprived males over the control males.

Porter, Webster, and Tickilder (1951) studied the influences of age and food deprivation on the hoarding behavior of the rat. Their results indicated that the number of pellets hoarded increased

linearly with the logarithm of the animals' age. Porter, Webster, and Licklider claimed that they obtained a significant amount of hoarding without depriving their rats. Waddell (1951) compared his hamster data to Porter's et al. non-deprived rat data. Allowing for the difference in body weight between the rat and hamster, Waddell found that the hamster hoarded approximately four and one-half times as much as the rat.

Environmental factors

A number of environmental factors have been shown to be of importance for hoarding. McCleary and Morgan (1946) found that low temperatures would produce hoarding in the rat. In fact there appeared to be an inverse relationship between the number of pellets hoarded and the ambient temperature. Smith and Ross (1953b), however, found that temperatures below 59° F. inhibited hoarding in the mouse.

Familiarity with the home cage has been shown to be an important factor in the hoarding behavior of the rat (Vlek and Miller, 1944). They found that rats would not hoard pellets in a strange cage. Miller and Vlek (1944) reported that the rats "familiarity" with the cage appeared to be based on olfactory rather than visual cues.

Stellar, Hunt, Schlosberg, and Solomon (1952) examined the effects of illumination of the hoarding alley. They hypothesized that lighting the alley would magnify the difference in hoarding between weanling food-deprived rats and normals. They believed that since light is an aversive stimulus for rats, lighting the runway would inhibit the normals but have less effect on the presumably more

motivated experimentals. Much to their surprise both groups hoarded more, although the controls were still inferior to the infant deprived group. The authors related their results to Sindra's (1948a) "security" hypothesis. Sindra found that rats that were non-hoarders in an enclosed runway situation would hoard in an open runway. Sindra's explanation was that non-hoarders in the closed runway felt secure and would remain at the end of the runway to eat. On the open runway, however, the rat felt insecure and would return to the home cage with the pellet, instead of eating it on the spot.

Emotionality

Hess (1953) studied "shyness" in rats as a factor in hoarding. The experimental situation was similar to Sindra's. Hess defined shy rats as those which would not enter an open runway for at least twenty minutes. Non-shy rats were those which would enter the runway in five minutes or less. Non-shy rats hoarded more, using the open runway, but this is not surprising since the hoarding trial was only one-half an hour long. However, the shy rats hoarded more pellets in a closed runway than non-shy rats. Hess mentions that his results confirm Sindra's (1948a). This is interesting since their results appear to be opposite. Apparently Hess's shy rats were more "insecure" than Sindra's. Stamm (1955) took the opposite tack and studied hoarding and aggressiveness in a paired fighting situation and attempted to correlate the results of this test with hoarding scores. He failed to find a significant correlation between aggressiveness and hoarding. Smith and Powell (1955) attempted to correlate measures of emotionality

with the hoarding behavior of mice. They measured three strains of mice on variables such as latency of movement into an open field, latency of movement into the hoarding alley, and defecation in the open field. The intrastrain analysis of these variables and the amount hoarded by each strain failed to reveal a significant relationship, although there did appear to be consistent relationships within the strains. For example, the S albinos overall had the longest latencies of movement, the highest defecation and urination scores, and also the largest number of pellets hoarded. Within the S albino strain, however, the relationship is reversed, the better hoarders are those animals with low latency scores.

Strain differences in hoarding

The above studies appear to indicate that strain differences in emotionality are important in the hoarding behavior of the rat and mouse. Stamm (1954a) has demonstrated specific strain differences in the rat. Stamm found that black-hooded rats were significantly superior to Irish and brow-hooded strains on various measures of hoarding behavior. Stamm found that the black-hooded rats had lower starting latencies, collected more pellets, and maintained hoarding in the post-deprivation period. Stamm (1956) selectively bred high and low hoarding strains. The F1 generation had hoarding scores similar to the high hoarding strain. When the F1 generation was backcrossed to the low-hoarding strain, bimodal hoarding scores were found in the backcross generation. This suggested that hoarding might be primarily influenced by one gene (Stamm, 1956). The genetics of

hoarding have also been studied in the mouse (Lindzey and Manosevitz, 1964, Manosevitz, 1965). Biometrical analysis of a selective breeding experiment (Manosevitz and Lindzey, 1967) showed that hoarding in the mouse is influenced by genes acting additively.

Experiential factors

Holland (1954) showed that prior experience in the hoarding alley affected the later hoarding behavior of rats. Holland's results indicated that rats allowed to explore a hoarding alley containing food showed improved hoarding when later tested against rats allowed to explore empty alleys. Smith and Ross (1953a) found that raising mice on a liquid diet did not interfere with later hoarding, contrary to Wolfe's (1935) results in the rat. Bevan and Grodsky (1958) raised hamsters under four different conditions. They found that hamsters raised on a liquid diet with litter on their cage floor showed the greater deficit in adult hoarding. The remaining groups showed a decreased deficit in hoarding in the following order: liquid diet and wire mesh floor, pellets and littered floor, pellets and wire mesh floor. These results would seem to indicate that the opportunity to manipulate pellets is important for adult hoarding in the hamster.

Physiological factors

Attempts have been made to determine the physiological bases of hoarding. Steller (1943) administered insulin, glucose, and epinepherine to rats in an effort to alter the hunger state of the animals. If hoarding is related to hunger such manipulations would be expected to influence hoarding. Neither insulin or glucose seemed

to effect hoarding, however, though epinepherine appeared to suppress it. Stellar (1951) tried to determine if alterations in metabolic rate would affect hoarding. Although thyroidectomy, thiouracil treatment, and thyroxine injections produced the expected changes in metabolic rate, none of the treatments had a significant effect on hoarding. Smith, Krawdyun, Ross and Wisehaupt (1954) adrenalectomized hamsters and found that it reduced their hoarding activity.

Zubeck (1951) reported that lesions anywhere on the cortex of the rat increased its hoarding. Stamm (1953), however, found that cortical lesions produced a deficit in hoarding. Stamm did not find any significant relationship between the degree of deficit observed and the size of the lesion, general location, subcortical damage, or preoperative hoarding. Stamm did, however, find a correlation between the hoarding deficit and damage to the midline of the rat's brain. Stamm (1954b) placed lesions in the median cortex. These lesions produced a significant reduction in hoarding behavior, although lateral cortical lesions did not. Similar results have been obtained in the hamster following median cortex lesions (Bunnell and Pinder, 1964).

Explanations of hoarding behavior

Several hypotheses concerning the nature of hoarding have been proposed. Morgan (1947) simply identifies hoarding as an instinctive behavior. The behavior is triggered by the crossing of a physiological threshold induced by deprivation. This is the so-called deficit hypothesis (Morgan, Stellar, and Johnson, 1943). Morgan, et al. (1943)

observed that removal of the rat's hoard did not seem to effect the behavior. They concluded that the goal of hoarding, once initiated, was the activity of hoarding itself. Miller (1945) was able to confirm Morgan, Stellar, and Johnson's (1943) results. Miller and Viek (1950) tested rats in an alley with five food bins at varying lengths from the home cage. The rats did not preferentially hoard from the food bin nearest the home cage, but instead hoarded equally from all five bins. Miller and Viek concluded that this inefficient pattern of hoarding indicated that the goal of hoarding in the rat is the activity itself, as Morgan, Stellar, and Johnson (1943) hypothesized.

Bindra (1948a) was unable to demonstrate changes in hoarding following deprivation of specific components of the rat's diet. Bindra rejected the deficit hypothesis and went on to develop the security hypothesis, already discussed. Bindra (1959) discusses his earlier results in terms of the shyness of the rats used, but neglects the findings of Hess (1953), who obtained opposing results, using an open runway similar to Bindra's (1948b). Although it would appear reasonable that emotional factors such as shyness might interact with environmental factors to effect hoarding in the rat, Bindra's security hypothesis so far has been useful only as a post hoc explanation for unexpected results (i.e., Stellar et al., 1952).

Marx (1950a) presumes that hoarding is learned. He considers hoarding to be the chaining of a number of independently learned acts, such as pellet seizing and carrying. These independent motor acts are integrated by the primary reinforcement of food and some possible

secondary reinforcers such as jaw movements. Marx (1950b), Marx (1951), Marx (1957), and Marx and Brownstein (1957) gathered evidence to demonstrate that the pellets hoarded functioned as primary reinforcers for hoarding activity. The results of these studies indicated that deprivation of pellets hoarded under varying levels of drive had little effect on the hoarding behavior of the rat, except under conditions of high-drive where an interference took place. Marx and Brownstein (1957) concluded that the terminal reinforcement of the hoarded pellets was unnecessary for the integration of hoard-behavior. Marx apparently has not attempted any further analysis of hoarding in learning theory terms.

All in all, most of the studies of hoarding behavior in the rat or mouse are of dubious relevance for hamster work. It has been mentioned that deprivation is unnecessary to initiate or maintain a high-level of hoarding in the hamster. Indeed, most hamsters will hoard on the very first trial though they have never been in the runway before. Although there is considerable variability in the amount hoarded from hamster to hamster, most tend to hoard from two to four times their body weights over a twenty-minute test period.

The key to the hamster's hoarding behavior lies in the fact that he is a hibernator. Hibernators such as the ground squirrel and woodchuck store body fat prior to hibernation (Lyman and Chatfield, 1955). The golden hamster, European hamster (Lyman and Chatfield, 1955), and the giant rat-headed hamster (Loukashkin, 1944) store food before they enter hibernation. Lyman (1954) has shown that hamsters with large

stores of food in their cages tend to enter hibernation significantly faster than animals with just a daily ration of food. Lyman (1954) concludes that the hamster needs to hoard to hibernate. The hamster frequently awakens from hibernation in order to eat, after which he resumes hibernation (Lyman, 1948). It would appear then that the hamster hoards to prepare for future hunger during hibernation. The hamster hibernates when exposed to low temperatures, but hoards at higher temperatures in preparation for this state.

Ventromedial Hypothalamic Nuclei, Feeding and Hoarding Behavior

The hoarding behavior of the hamster would appear to be food-motivated behavior. One area of the nervous system which has been shown to be important in feeding behavior is the ventromedial portion of the mammalian hypothalamus. Although the relationship of the ventromedial nuclei to hoarding behavior has not been previously investigated, there is a considerable body of literature concerning their role in feeding behavior.

The question of hypothalamic involvement in ingestive behavior was first raised by the obesity seen in humans suffering from pituitary tumors. The changes produced by these tumors became known as Frohlich's Syndrome. Frohlich (1940) felt that the primary cause of the obesity was pituitary damage. Rosenzweig (1963) has reviewed the controversy between Frohlich and Erdheim, who believed that the obesity was produced by hypothalamic injury. Although many experiments tended to support the idea that hypothalamic damage was the primary cause of the

obesity, it was not until the development of the stereotaxic technique that the question was resolved. Using the stereotaxic method, Hetherington and Ranson (1940) showed that damage to the ventromedial hypothalamic nuclei appeared to produce obesity.

The question then arose as to the primary effect of the lesion. Hetherington and Ranson (1942) suggested that the lesion produced a metabolic change such that the animal was unable to utilize its body fat. Other researchers (Brobeck, Tepperman and Long, 1943; and Tepperman, Brobeck and Long, 1943) were unable to find any clear changes in metabolism following ventromedial lesions. Examination of the oxygen consumption and respiratory quotients of ventromedial lesioned rats failed to show any significant changes directly attributable to hypothalamic injury. In addition, ventromedial lesioned rats could apparently lose weight when deprived of food, and did not become obese when restricted to normal food intake (Brooks, 1946; Brooks and Lambert, 1946). Brooks, Lockwood, and Wiggins (1946) reported essentially similar results. Brooks, Merrine, and Lambert (1946) also indicated that the food-feces ratio, a measure of intestinal absorption, was normal in ventromedial rats.

The inability of these researchers to discover metabolic deficits in ventromedial rats led to attempts to investigate the feeding patterns of these animals. Brobeck, Tepperman, and Long (1943) introduced the phrase "hypothalamic hyperphagia" to describe the ingestive behavior. They also observed that the hypothalamic animals showed two distinct phases of food consumption. The first is the "dynamic" phase occurring

immediately or very soon postoperatively. During the dynamic phase the animal typically eats two to three times as much as normal and gains weight rapidly. Eventually the animal's food intake decreases towards preoperative levels and the animal's weight levels off and remains stable, but at a high level. This is referred to as the static phase.

Since there appeared to be no metabolic deficits in hypothalamic hyperphagics, most studies, until very recently, tended to focus on the feeding behavior of the hyperphagic animal. Kennedy (1950) made the important discovery that obese ventromedials decreased their food intake and lost weight when their food was adulterated with kaolin. Adolph (1947), however, had shown that normal rats will increase their intake when their food is diluted with some inedible substance. Miller, Bailey, and Stevenson (1950) found a similar result with obese rats that presumably had received ventromedial lesions. They found a decrease in food consumption after addition of quinine to the animal's diet. Teitelbaum (1955) made a more systematic study of this effect. He found that the addition of as little as 25% cellulose or 0.125% quinine produced almost total cessation of eating in obese hypothalamic hyperphagics. Dynamic hyperphagics were little effected, and did not fall to the intake level of normals until 75% adulteration of their food with cellulose, nor were they significantly affected by quinine. On the other hand, normals decreased their intake with a 50% dextrose diet, while obese hypothalamics overate on this diet. Dynamic hyperphagics continued to eat the same quantity of food, which in this case amounts to an increased caloric intake. Teitelbaum concluded that

obese hyperphagic rats were sensitive to the stimulus characteristics of their diet.

Miller, Bailey, and Stevenson (1950) had shown that in addition to a decrease in food consumption, hypothalamic lesioned rats showed deficits on certain tasks designed to measure motivation. On tasks such as lever pressing, pushing a weighted lid off a jar, latency in running a runway, strength of pull on a harness, the hypothalamics were deficient. All of the tasks used food as a reward and only on approach to a food cup protected by an electrified shield did the hypothalamics show no significant differences from normals. They interpreted the data to mean that although hypothalamic rats showed increased food consumption the rats were actually not as motivated as normal animals to obtain food. Teitelbaum (1957) studied the random and food-directed activity of hyperphagic rats. He found that the random activity of both dynamic and obese hypothalamic was lower than normals. Teitelbaum also showed that the obese animals showed very poor performance on a fixed ratio bar pressing schedule. The dynamic hyperphagics were also inferior to normals, but were markedly superior to the obese rats. Teitelbaum (1961) stated that motivation is weaker in the hyperphagic rats even though they overeat.

Teitelbaum and Campbell (1958) investigated the feeding patterns of hyperphagic rats. On a liquid diet hypothalamic rats do not eat more often than normals, nor do they eat any faster. Hyperphagic rats do eat for a longer time once they have begun; however, on a solid diet they eat more frequently. Teitelbaum and Campbell (1958) suggest that

more frequent eating on the solid diet is probably due to the fact that the greater bulk of a solid diet prevents the animal from obtaining as many calories per meal. This would actually demonstrate that the hyperphagics are regulating their calories. Williams and Teitelbaum (1959) presented further proof of caloric regulation. By diluting a liquid diet with water they demonstrated that both normal and hyperphagic rats would increase the number of meals they ate to maintain a constant caloric intake.

The results of these studies leave us with a rather peculiar picture, best described by Teitelbaum (1961). He describes the hypothalamic hyperphagic as an animal that overeats but does not show increased hunger, who regulates its calories but is hypersensitive to the palability of the diet. It is questionable that the data on ventromedial lesioned rats will in fact support the idea that they are truly less motivated for food. Both Falk (1961) and Reynolds (1965) have criticized the concept of lowered motivation in the ventromedial rat, pointing to certain deficiencies in the Miller, Bailey, and Stevenson (1950) study. One of the first difficulties with the Miller et al. study was the fact that only four out of eleven operated rats showed the primary characteristic of hypothalamic hyperphagia, that is, increased food intake and concomitant obesity on ordinary lab chow. Although these animals did eventually become obese on a palatable high-fat diet, it is possible that their lesions were comparable to those placed more laterally in the ventromedial nuclei and produced finicky, non-obese animals (Greff and Stellar, 1962). It is impossible to say

anything about the exact locus of the lesions in this study because no histology was presented. Most of the tasks used by Miller and his co-workers were strongly confounded with the activity level of the animal as is the fixed ratio bar press schedule used by Teitelbaum (1957). Falk (1961) points out that the ventromedial animal must be considered a low-activity preparation. Hetherington and Ranson (1942), Brooks (1946), and Teitelbaum (1957) have all found lowered activity in both dynamic and obese hyperphagics. Gladfelter and Brobeck (1962) found that damage to the medial hypothalamus between the preoptic and premammillary area produced hypoactivity in the rat. The hypoactivity appeared to be independent of other effects of the damage. Falk (1961) has examined the bar pressing activity of hypothalamic hyperphagics in the dynamic phase. Falk used a variable interval 1' schedule instead of a fixed ratio schedule. He found that the ventromedials increased their response rate postoperatively. He also found a significant correlation between the increase in responses and the weight eventually attained by the animal. The results of this study do not seem to be compatible with the claims of lower motivation in hypothalamic hyperphagic rats.

It would also appear that the increased sensitivity to changes in palatability of food observed in obese hypothalamic rats is due to their obesity. Kennedy (1950) reported that older, naturally fat rats also show a decrease in food intake if kaolin is added to their diet. Similarly, Teitelbaum (1955) concluded that it was the obesity of the static hypothalamic rats which was responsible for their

hypersensitivity to stimulus properties of the diet. The results of the dynamic hyperphagics in Teitelbaum's study are not at all in agreement with a hypothesis of lowered motivation for food. Miller (1955) has added to the confusion by stating, 'Under somewhat different conditions which are not yet well understood by us the rats with hypothalamic lesions cannot only eat more, but also work harder for food."

Hyperphagia and obesity have been reported in several species following ventromedial hypothalamic lesions. Mayer, French, Zighera, and Barnett (1955) reported on the mouse, Hamilton and Brobeck (1964) on the monkey, Wheatley (1944), and Skultety (1966) on the cat. All of these species seem to show a dynamic and static phase similar to that observed in the rat. Few studies investigating the food-motivation behavior of ventromedial lesioned animals other than the rat have been performed. Hamilton and Brobeck (1964) found no deficits in hypothalamic monkeys tested with quinine adulteration or on a fixed ratio schedule, also the lesioned animals increased in dominance in a food motivated situation but not in dominance test using shock motivation. Anliker and Mayer (1956) tested hypothalamic mice on an FR 25-1 schedule. These animals did not exhibit any impairment of performance.

Although there are several theories of the role of the ventromedial nuclei in the regulation of hunger, only the two major theories will be discussed at this time. These two theories are known as the "glucostatic" and the "lipostatic" theories. Mayer (1955) is the author of the glucostatic theory. The core of this theory is that

hunger will be correlated with utilization of glucose by body cells, Mayer (1955) suggests that the arterio-venous glucose differences should be related to hunger. When the difference between A-V glucose levels is large considerable glucose is available to the body cells, and consequently, the organism should be sated. When A-V differences are low little glucose is available and the animal should feel hungry Mayer (1955) indicates that manipulation of the A-V glucose level supports the above assertions. Mayer (1955) also postulates the existence of glucoreceptors in the ventromedial area. These would respond to the level of glucose in the blood and regulate feeding accordingly. Initially, the destruction of the ventromedial nucleus in mice by goldthioglucose injection (Marshal, Barnett, and Mayer, 1955) was taken as evidence for glucoreceptors. Liebelt and Perry (1957) showed that destruction was present throughout hypothalamus and fornix of the rat following GTG injection, so the effect may be very specific to mice Using electrophysiological techniques Anand, Dua, and Singh (1961) and Anand, China, Sharma, Dua, and Singh (1964) have shown that neurons in the ventromedial area of cats respond with increased firing rates with high A-V glucose differences and lowered firing with decreased A-V differences. Although their results are compatible with the glucostatic theory, Cross (1964) has criticized this study on the grounds that the blood-glucose levels produced were 300-400% higher than normal. Cross doubts that such gross manipulations bear any relationship to normal physiological response At present it would appear that the existence of glucoreceptors has neither been conclusively proven or disproven.

The second major theory dealing with regulation of hunger is the so-called lipostatic theory first proposed by Kennedy (1953). This theory postulates the existence of some metabolite related to fat deposits to which cells located in the ventromedial area are sensitive. Hervey (1959) using parabiotic rats showed that when one of the pair becomes obese after a hypothalamic lesion, the other animal eats less and becomes very thin. Presumably the normal animal was detecting the increased fat deposits and its feeding behavior was correspondingly inhibited. Haessler and Crewford (1965) obtained similar results with a parabiotic study of genetically obese mice. A dissenting note was added by Han (1964), who was unable to replicate Hervey's study. Attempts to identify the circulating metabolite have been unsuccessful. Hoebel and Teitelbaum (1966) have also presented evidence indicating that the rat regulates its food intake on the basis of its fat stores. Hoebel and Teitelbaum produced hyperphagia and subsequent obesity by means of insulin injections. Rats made obese in this fashion became anorexic until their weight fell to normal following cessation of insulin treatment. If insulin obese rats received ventromedial lesions, then little hyperphagia and weight gain was observed. Clearly the normal rat regulates its food intake on the basis of weight; ventromedial lesions remove his ability to perform this function and the animal moves to a higher weight level. However, as Hoebel and Teitelbaum (1966) show, he still regulates his weight, for if the rat is force-fed so that his weight is driven even higher, he will become anorexic and will drop back to the level of obesity attained in the

static phase. Precisely how the obese hypothalamic animal regulates his weight remains a mystery; certainly the ventromedial hypothalamic nuclei cannot be the only areas of the brain sensitive to fat deposits.

Although only two theories concerning hypothalamic regulation of food intake have been presented here, Anand (1961) points out the importance of other factors such as temperature, gastric distention, and learning on the regulation of feeding.

The Septal Nuclei, Hoarding and Feeding Behavior

In general there appears to be a paucity of information concerning the effects of septal lesions on hoarding behavior. Septal lesions placed in two hamsters appeared to produce a deficit in hoarding (Bunnell and Metalka, unpublished). It would appear necessary to test the effects of septal lesions on the hoarding behavior of a larger number of animals before any conclusions can be drawn on the role of the septal nuclei in hoarding behavior

Septal lesioned hamsters commonly become very obese (Sodetz, 1965). This suggests a possible relationship between the septal forebrain and ventromedial hypothalamic areas. As yet, no one appears to have investigated limbic-hypothalamic anatomical connections in the hamster. A review of the literature reveals some disagreement between authors on septal-ventromedial connections. Studies examining the distribution of fornix fibers have been included, since post-commisural septal lesions almost invariably damage fornix fibers. Crosby and Woodburne (1951), Simpson (1950), Votaw (1960) all report some distribution of fornix fibers to the ventromedial nuclei of the

monkey. Sprague and Meyer (1950) report some terminal degeneration in the ventromedial nucleus of the rabbit following fornix damage in the rabbit. Degeneration in the ventromedial area could not be demonstrated following septal or fornix damage in the monkey (Valenstein and Nauta, 1959), cat (Nauta, 1958, Valenstein and Nauta, 1959), guinea pig (Valenstein and Nauta, 1959), or rat (Guillery, 1957, Nauta, 1956, Powell, 1963; Rasmsan, 1966, or Valenstein and Nauta, 1959). Nauta (1956) did report some degeneration in the periventricular system of the medial hypothalamus. Valenstein and Nauta (1959) reported a similar finding in the guinea pig. It would appear that connections between the septal and ventromedial areas are sparse, if in fact they even exist.

Some evidence for the functional relationship between septal and ventromedial nuclei was presented by Sutin (1963). Sutin found that responses of 5-7 msec. could be recorded from the ventromedial nucleus of the cat, following stimulation of the septal area above the anterior commissure. Stimulation of the hippocampus or fornix fibers did not produce responses in the ventromedial nucleus, indicating some specificity between the ventromedial and septal areas.

Although fornix damage (Ehrlich, 1963) and hippocampal lesions (Kimble and Coover, 1966) produce increases in food intake of the rat, the body weight of these animals does not appear to increase. It is likely that the food intake increase is secondary to the increased activity of these animals (Ehrlich, 1963). The development of obesity in the septal hamster appears to be a unique finding and the only evidence for a septal-ventromedial interaction in the hamster.

Amygdaloid Nuclei: Hoarding and Feeding Behavior

Evidence for the role of the amygdala in hoarding is equally as sparse as that for the septal area. Bunnell, Soldetz, and Shalloway (1965) reported that amygdala lesioned hamsters showed an increase in hoarding. However, these animals were tested in a social dominance situation and the apparent increase in hoarding might only reflect a disruption of social behavior. Amygdaloid lesioned hamsters appear to be less interested in the other test animal and the apparent increase in hoarding may only represent a shift of interest to inanimate compared to preoperative behavior. Amygdaloid lesioned hamsters should be examined in a situation specifically designed to test hoarding to determine if they will show an absolute increase in hoarding behavior. A relatively extensive survey of the literature failed to reveal any other attempts to examine the effect of amygdaloid lesions on hoarding.

Several studies have implicated the amygdala in feeding behavior. Anand and Brobeck (1952) reported aphagia in rats following amygdaloid damage. Green, Clemente, and DeGroot (1957) indicated that anterior amygdala lesions interfered with eating in the cat. However, they indicated that an increase in food intake occurred in cats with lateral and basal nuclei damage (Morgane and Kosman, 1960; Kolkagami, Fuse, Hiroki, Kazami, Kageyama, 1958; and Wood, 1958). Grossman and Grossman (1963) found transient

increases in food intake after small posteroventral amygdala lesions. Similar results were found in the dog following amygdaloid-hippocampal damage (Fuller, Rosvold, and Mirsky, 1957). Amygdaloid ablation produced hyperphagia in the monkey (Schwartzbaum, 1961). The tendency of amygdaloid lesions to produce increases in food intake would appear to suggest a relationship between at least certain of the amygdaloid nuclei and the ventromedial nuclei.

Nauta (1961) could not find degeneration in the ventromedial nuclei after ablation of the amygdala in the monkey. Ban and Omukai (1958), however, traced degeneration through the stria terminalis to the ventromedial hypothalamus of the rabbit, after damaging the medial and cortical amygdaloid nuclei. Adey and Meyer (1952), Adey, Rudolph, Kime and Harrit (1958) reported terminal degeneration in the ventromedial nuclei of the monkey following amygdaloid damage.

Sutin (1963) was able to record responses from the ventromedial nuclei of the cat, usually with a latency of 8 msec. after amygdaloid stimulation. In general, most evidence points to an intimate relationship between the amygdala and ventromedial nuclei. However, the lack of anatomical and electrophysiological data on the hamster makes prediction of food intake changes following amygdala damage difficult.

Purpose and Hypothesis

A review of the literature would seem to indicate that the septal, amygdaloid, and ventromedial nuclei all appear to play a role in ingestive behavior. Little evidence exists as to the role of these structures with respect to an unlearned food motivated behavior such as hoarding. The evidence collected in this laboratory could be interpreted to indicate a reciprocal relationship between the septal and amygdaloid areas with respect to hoarding. Possibly the septal area plays a facilatory role, with the amygdala acting as inhibitor of this behavior. It is also possible that these limbic areas act upon the hypothalamus, with regard to hoarding in much the same fashion that King (1958) has suggested that they act with respect to emotionality. Certainly they might interact with an area shown to be important in feeding such as the ventromedial nuclei of the hypothalamus. It would appear profitable to examine these neural areas to determine their respective roles in hoarding and feeding behavior of the hamster. The results of such an investigation might also shed some light on possible limbic-hypothalamic interactions with respect to hoarding and feeding.

Some general hypotheses can be developed on the basis of the studies reviewed. Septal forebrain lesions might be expected to decrease hoarding, while amygdaloid lesions may increase it. The apparent obesity of septal animals would suggest that they will show an increase in food intake. Both hyperphagia and aphagia have

been reported following amygdaloid lesions, so it is difficult to hypothesize about the effects of amygdaloid lesions on the food intake of the hamster. Anand and Brobeck (1952) have reported mild aphagia in the rat. The hamster, being a rodent, might be expected to respond in a similar manner and show some degree of aphagia after amygdaloid lesions.

Ventromedial hypothalamic damage has apparently produced hyperphagia and concomitant obesity in every species studied. Ventromedial hypothalamic damage might be expected to produce the same effect in the hamster. If ventromedial lesions do in fact decrease food motivated behaviors, hoarding is also likely to be decreased.

METHOD

Subjects

Forty-five male Syrian golden hamsters were used in this study. All were randomly bred stock obtained from Manor Farms, Staatsburg, New York. The animals were divided into three groups designated H-I, H-II, and H-III. The animals were further divided into three lesion groups: a septal group designated as S, an amygdaloid group designated A, and a ventromedial hypothalamic group designated V. Three electrode insertion groups were also run, these consisted of animals who had electrodes inserted in the septal, amygdaloid, or hypothalamic areas and withdrawn without current being passed. These were respectively a ventromedial hypothalamic control group designated VC, a septal control group designated SC, and an amygdaloid control group designated AC. A normal control group of eight hamsters designated HC was also run. Four animals of this group had muscle retracted from their skulls, the remaining four received anesthetic doses of nembutal. The distribution of the lesion and control animals over the three experimental groups is shown in Table I.

The animals of group H-I were approximately six months old at the initation of testing and twelve months old at the cessation of testing. Group H-II animals were four months old at the initiation of testing and nine months old at the cessation of testing. Group H-III animals were three months of age at the initiation of testing and six months old at the end.

Table 1

Distribution of the Experimental and Control Animals Among the Three Main Experimental Groups.

Experimental Groups	S	A	V	SC	AC	VC	NC
H-I	1	-	5	-	-	2	2
H-II	5	6	-	2	2	-	2
H-III	2	4	2	2	2	2	4
Total n's per group	8	10	7	4	4	4	8

S = Septal Group

A = Amygdala Group

V = Ventromedial Hypothalamic Group

SC = Septal Control Group

AC = Amygdala Control Group

VC = Ventromedial Hypothalamic Control Group

NC = Normal Control Group

Apparatus

Several batteries of hoarding cages were used. Each cage consisted of a living cage (6" x 8" x 7½"). The cages were constructed of wood and one-fourth inch hardware cloth. The cages were painted white or grey. A guillotine door opened from each living cage into a one-fourth inch hardware cloth runway. The runway was twenty-four inches in length and three inches square in cross section. The animals hoarded Purina Lab Chow pellets from a removable one-fourth inch hardware cloth basket placed at the end of the runway. These baskets could hold approximately twenty-five pellets. Wooden doors at the far end of the runway gave the experimenter access to the baskets. The runways were illuminated by four 100 watt light bulbs placed on a board suspended approximately three feet above the runway.

Daily water intake scores were obtained using Curtin CC 112 measuring tubes.

Behavioral Procedures

Hoarding trials

All animals received twenty daily preoperative and fifteen daily postoperative hoarding trials. All animals lived in the hoarding apparatus for the duration of the hoarding test phases of the experiment. During this period they were confined to the living cage part of the apparatus, except for hoarding trials when they were released into the runways. A hoarding trial consisted of a twenty-minute period during which the hamster was allowed to collect pellets

from the wire baskets placed at the end of the runway. The baskets were refilled if emptied before the end of the test period. At the end of the twenty-minute test period the hamster was locked in the living cage, the pellets hoarded and the animal were removed and both its both weight and pellet weight were recorded.

The first ten preoperative trials were conducted under ad lib feeding conditions (the daily food ration was four pellets). The uneaten portion of daily ration was removed prior to the hoarding trial. The 11th-14th preoperative hoarding trials were carried out under a deprivation schedule. On trials 11, 12, and 14, the animal was allowed to feed for one hour daily, immediately prior to the hoarding trial. Between the 11th and 12th trials, Ss were allowed to eat one 3-5 gram pellet. This was done to check the rapid weight loss seen at the initiation of deprivation and to prevent inanition. The animals were returned to ad lib feading for the 15th-20th preoperative hoarding trials.

At the end of the preoperative hoarding tests, the animals were placed in individual cages and returned to the main colony room. Here food and water intake was measured and nest construction was rated, prior to operation. Following postoperative measures of food, water and nest building, the hamsters were returned to the hoarding cages and received 15 hoarding trials. These trials were conducted exactly as the preoperative trials. The first ten were conducted under ad lib feeding and the 11th-14th trials under deprivation. Following the postoperative hoarding tests the animals were again

returned to the colony room to obtain further intake and nesting scores.

Food and water intake

Since there is some variation in the number of pre- and postoperative intake scores between the groups, they will be described separately.

Group M-1: -- A total of ten daily food and water intake scores were obtained preoperatively. Powdered Purina Lab Chow was used for the food intake, tap water for the water intake. Group M-1 animals were also tested on a General Biochemical Hamster diet, in general they did not appear to find this diet particularly palatable and further use of this diet was discontinued, following postoperative testing of this group. Postoperatively the animals of this group had difficulty in maintaining body weight. Only six food and water intake scores were obtained postoperatively since most of the time the lesioned animals had to be maintained on a palatable wet mash diet.

Group M-11: -- Five consecutive days of food and water intake were obtained preoperatively in all the animals of this group. Some of the amygdaloid lesioned animals had difficulty in maintaining body weight postoperatively and had to be placed on a wet mash diet. At least ten daily food and water intake scores were obtained from these animals postoperatively, however. Fifteen daily food and water intake scores were obtained from all other animals in this group.

Group N-III -- Ten preoperative daily food and water intake scores were obtained from this group. A total of fifteen postoperative food and water intake scores were obtained from each of the animals in this group.

Nesting scores

The quality of the nests constructed by the animals used in this experiment was rated on a 0-7 point rating scale. A copy of this scale is presented in Appendix A. Some animals were independently rated by two observers to obtain reliability data.

No preoperative ratings were obtained on Group N-I, but five postoperative ratings were obtained. The animals of groups N-II and N-III received five preoperative and ten postoperative ratings.

Surgical and Histological Procedures

All surgery was performed under pentobarbital sodium (nembutal, 60 mg/kg). Following incision of the scalp the fronto-nasal and lambda sutures were leveled and all anterior-posterior coordinates were measured from bregma. Trephine holes were drilled in the skull and the holes widened if necessary with a corneoscleral punch, to permit bilateral insertion of the electrode.

The coordinates used for the septal lesion were 6.5 mm. anterior to bregma, 0.5 mm. lateral to midline, and 4.0 mm. ventral to the cortex. The coordinates for the amygdaloid lesion were 3.5 and 4.5 mm anterior to bregma, 4.0 mm. and 6.5 mm. ventral to the

cortex. The ventromedial hypothalamic coordinates were 3.5 mm. anterior to the bregma, 0.5 mm. lateral to midline, and 7.5-8.0 mm. ventral to cortex. Appropriate electrode insertion control groups used the same coordinates as their respective experimental group.

All lesions were produced stereotaxically by stainless steel electrodes insulated except for the tip. The animals were held in a Kopf stereotaxic instrument and the lesions were produced by a Grass RF lesion generator.

Upon completion of data collection the operated and electrode insertion groups were given a lethal dose of nembutal and perfused with a 10% formalin solution. Their brains were removed and embedded in celloidin. The lesioned and control brains were sectioned at 30 micra. Every fourth section was stained with Cresyl Violet and mounted for reconstruction of the lesion. Animals who had muscle retractions were given a lethal dose of nembutal and were examined to determine if abnormal scar tissue was present.

RESULTS

Hoarding Under Ad Lib Feeding

The total weights of pellets hoarded over the ten preoperative and ten postoperative trials under ad lib feeding are presented in Appendix B. The means for each group are presented in Table 2.

A Kruskal-Wallis test (Siegel, 1956) did not reveal any significant variations in the preoperative scores of the various groups. An H value of 23.2, significant at the .001 level, was obtained from a Kruskal-Wallis test of the postoperative scores.

Further statistical analysis was necessary to determine which groups showed significant variations. A Newman-Keuls test (Winer, 1962) was adopted for this purpose. Winer (1962) indicated that the number of subjects should not be too diverse from group to group. Since the number of subjects ranged from four in the operated control group to ten in the amygdala group, the ventromedial, amygdala, and septal control groups were combined to form an operated control group, designated OC. The pooling of the three groups was legitimate since a Kruskal-Wallis test failed to reveal a significant difference between them ($H = 3.12$ $p > .104$). A Newman-Keuls test was then computed between the five groups. The results of this test are presented in Table 3. The septal animals differed from both the operated and the normal controls at the .05 level. Similar

Table 2

Pre- and Postoperative Mean Weight (Gms.) of Pellets Hoarded per Trial under Ad Lib Feeding and Deprivation Conditions

	Ad Lib Feeding		Deprivation	
Group	Preop	Postop.	Preop	Postop.
Septal n = 8	363.1	24.2	434.5	46.6
Amygdala n = 10	278.7	144.4	381.3	171.1
Ventromedial Hypothalamics n = 7	225.6	18.1	396.1	67.1
Operated Controls n = 12	290.4	203.1	477.6	353.4
Normal Controls n = 8	243.1	220.4	378.3	273.3

Table 3

Newman-Keuls Test on Postoperative Mean Total Weight (Gms.) of Pellets Hoarded Under Ad Lib Feeding

	V	S	A	OC	NC
Means	181.0	242.0	1443.3	2031.0	2204.0
V 181.0	-	61.0	1262.0	1850.0*	2023.0*
S 242.0		-	1201.0	1789.0*	1962.0*
A 1443.0			-	588.0	761.0
OC 2031.0				-	173.0
NC 2204.0					-

* P < .05

	r = 2	r = 3	r = 4	r = 5
Critical Values				
d.f = 40				
.05 Level	1338.0	1609.0	1773.0	1890.0
.01 Level	1787.0	2045.0	2199.0	2307.0

V = Ventromedial Hypothalamic Group n = 7

S = Septal Group n = 8

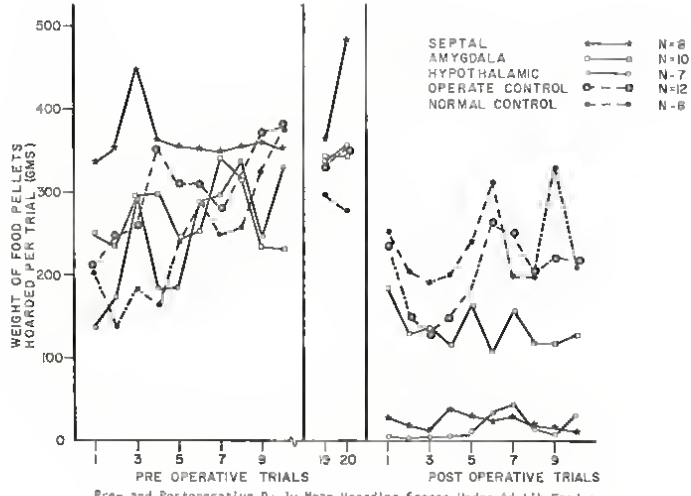
A = Amygdala Group n = 10

OC= Operate Control Group n = 12

NC= Normal Control Group n = 8

results were obtained for the ventromedial group. An examination of the postoperative mean per trial of each group, also shown in Table 2, indicated that the septal and ventromedial groups showed significantly lower hoarding scores. The Newman-Keuls test did not reveal a significant difference between the amygdala group and any of the groups. In an effort to clarify the position of the amygdala group a series of Mann-Whitney U tests (Siegel, 1956) were performed between the amygdala group and the experimental and control groups. The amygdalas did not differ significantly from the control groups. A significant difference ($n = 8$, $n = 10$, $U = 8$, $P < .02$) was found between the septal and amygdala groups. A significant difference was also found between the amygdala and ventromedial groups ($n = 7$, $n = 10$, $U = 6$, $P < .002$). Although this procedure is questionable, the results of the Mann-Whitney tests seem to indicate that though the amygdalas suffered a postoperative reduction in hoarding they did not differ from the controls.

The daily mean hoarding scores for the pre- and postoperative trials under ad lib feeding are presented in Figure 1. Trials 1-10 are shown followed by the 19th and 20th trials. Trials 19 and 20 are included to illustrate the preoperative post-deprivation hoarding trials. An examination of Figure 1 indicates that the ventromedial and septal animals show a clear decrement in hoarding postoperatively. The amygdala group was slightly lower than the control groups. There was no apparent upward trend in the daily hoarding scores of the experimental groups during the postoperative testing period.



Pre- and Postoperative Da ly Mean Hoarding Scores Under Ad Lib Feeding

Inspection of Table 2 and Figure 1 indicates that all groups showed a reduction of hoarding over the postoperative period. Computation of a Wilcoxon Matched Pair Signed Ranks Test (Siegel, 1956) between the pre-and postoperative hoarding scores of the animals in each group failed to show a significant reduction in either of the control groups. Significant reductions were found for all of the experimental groups, the amygdalas showed significant drop ($T = 7$, $P < .05$), the septals ($T = 0$, $P < .01$), and the ventromedials ($T = 0$, $P < .02$). Again the amygdala group appears to occupy an ambiguous position. Although the results of statistical tests between the postoperative hoarding scores of the groups indicated they were not different from normals and operated controls, the amygdala group did show a significant drop from their preoperative values.

Hoarding Under Deprivation Conditions

The deprivation schedule used for pre-and postoperative testing produced a decrease of approximately 5-10% in body weight. The weight of pellets hoarded over the pre-and postoperative deprivation trials are shown in Appendix C. A Signs test (Siegel, 1956) indicated the preoperatively, deprivation produced a significant increase in hoarding ($z = 2.4$, $P < .01$). This was tested using the preoperative ad lib scores from Appendix B and preoperative deprivation scores from Appendix C.

The approach used for statistical analysis of the deprivation data was identical to that used for the analysis of the ad

lib scores. A Kruskal-Wallis test on the preoperative hoarding scores did not reveal a significant difference between the groups. A significant H value was obtained between the postoperative total weight hoarded ($H = 22.9$, $P < .001$). As with the postoperative ad lib scores the VC, AC, and SC groups were tested with the Kruskal-Wallis test ($H = 4.96$, $P < .097$). This did not reveal a significant difference between the three groups so they were pooled to form a operated control group. The mean weight per trial for each group is shown in Table 2. The results of a Newman-Keuls test are shown in Table 4. The results are similar for those found under postoperative ad lib feeding condition. The septals differ significantly from the operated control groups at the .01 level, and the normal controls group at the .05 level. The ventromedials differ from the normals at the .05 level and the operated controls at the .01 level. Once again the amygdales do not differ significantly from any of the experimental or control groups.

An examination of the postoperative mean weights hoarded per trial during deprivation, presented in Table 2, indicates that the amygdalas were again lower than the control groups. Once more a series of Mann-Whitney U tests was computed between the amygdales and the experimental and control groups. The amygdalas did not differ from the normal controls. Significant differences were found between the amygdalas and septals ($n = 8$, $n = 10$, $U = 4$, $P < .001$), ventromedials ($n = 7$, $n = 10$, $U = 4$, $P < .001$), and operated controls ($n = 7$, $n = 10$, $U = 4$, $P < .01$).

Table 4

Newman-Keuls Test on Postoperative Mean Total Weights (Gms.) of Pellets Hoarded Under Food Deprivation

		S	V	A	NC	OC
	Means	233.0	336.0	857.0	1367.0	1767.0
S	233.0	-	103.0	624.0	1134.0*	1534.0**
V	336.0		-	521.0	1031.0*	1431.0**
A	857.0			-	510.0	910.0
NC	1367.0				-	400.0
OC	1767.0					-

* P < .05
** P < .01

		r = 2	r = 3	r = 4	r = 5
Critical Values					
d.f. = 40					
.05 Level		766.0	922.0	1015.0	1083.0
.01 Level		1023.0	1171.0	1281.0	1321.0

S = Septal Group n = 8

V = Ventromedial Hypothalamic Group n = 7

A = Amygdala Group n = 10

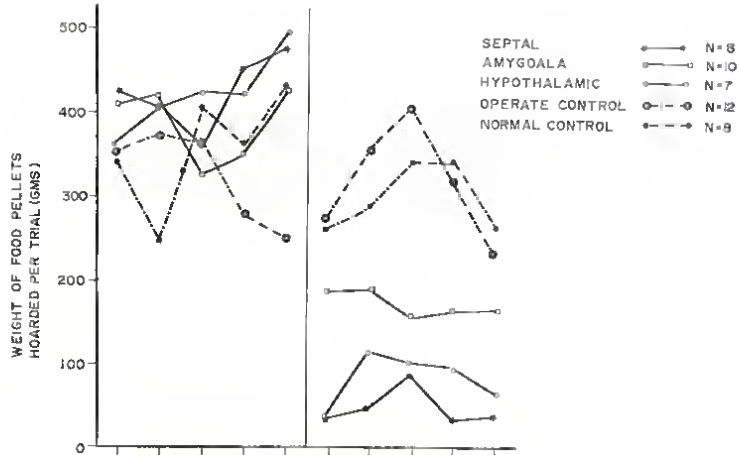
OC= Operate Control Group n = 12

NC= Normal Control Group n = 8

A further examination of the postoperative scores presented in Table 2 and Appendix C indicated that the experimental groups were relatively unresponsive to postoperative deprivation. The operated control group showed a higher mean score than the normal group which probably accounts for the significant U value between the amygdales and the operate controls.

Figure 2 shows the daily mean weights of food pellets hoarded during the pre-and postoperative deprivation trials. As with the ad lib conditions, the septal and ventromedial groups show the greatest decrement, with the amygdales occupying an intermediate position. The postoperative daily scores are somewhat below the preoperative values for the control groups as the overall means in Table 5 would indicate. The operated control group appear to exceed the daily means of the normal group. Again this would agree with the means shown in Table 2. There appears to be no indication of an upward trend in the hoarding of the experimentals over the five postoperative trials.

The results of the postoperative deprivation trials did not indicate that the hoarding behavior of the ventromedial and septal groups was significantly effected. There appeared to be no tendency for the behavior to rise to the level of the control animals during this period. Comparison of the pre-and postoperative mean weight of pellets hoarded per deprivation trial indicated that there was a general reduction in postoperative hoarding. Wilcoxon Matched Pair Signed Ranks tests were computed for each group. The results of these tests were very similar to those found between pre-and postoperative ad lib conditions. Neither of the control groups showed a significant



Pre- and Postoperative Daily Mean Hoarding Scores Under Food Deprivation

decrease in postoperative hoarding. The treatment groups all showed a significant reduction compared with the preoperative deprivation scores. Amygdales showed a significant reduction ($T = 4$, $P < .02$), septals ($T = 0$, $P < .01$), and ventromedials ($T = 1$, $P < .05$). Again the amygdales appear to show a decrement in postoperative hoarding but the drop is not significantly less than that experienced by the control groups

Food and Water Intake

Table 5 shows the pre-and postoperative mean food intake scores for the various groups. The individual means are presented in Appendix D. A Kruskal-Wallis test of the preoperative scores did not reveal overall significance between the groups. A significant H value ($H = 18.6$, $P < .01$) was found after a Kruskal-Wallis test of the postoperative scores. As with the hoarding scores the AC, VC, and SC groups were pooled after a Kruskal-Wallis test failed to indicate a significant difference between the three groups ($H = 0.7$, $P < .104$). The results of a Newman-Keuls test on the postoperative scores are shown in Table 6. The septal group shows a significant increase over all other groups in postoperative food intake. The postoperative increase is 20% higher in the septals.

The increase in food intake shown by the septal group is reflected in an obvious weight increase illustrated in Figure 3. A Kruskal-Wallis test of the body weight reached by the various groups on the 15th postoperative day failed to yield a significant difference between the groups. A Kruskal-Wallis test of the amount

Table 5

Pre- and Postoperative Mean 24 Hour Food Intake (Gms.)

Group	Preop	Postop
Amygdala n = 10	10.2	6.9
Ventromedial Hypothalamic n = 7	9.7	8.5
Septal n = 8	9.3	11.2
Operate Control n = 12	9.3	7.7
Normal Control n = 8	9.7	8.8

Table 6

Newman-Keuls Test on Postoperative Mean Food Intake Scores (Gms.)

	A	OC	V	NC	S
Means	6.0	7.6	8.5	8.8	11.2
A 6.0	-	0.7	1.6	1.9	4.3**
OC 7.6		-	0.9	1.2	3.6**
V 8.5			-	0.3	2.7**
NC 8.8				-	2.4**
S 11.2					-

** P < .01

	r = 2	r = 3	r = 4	r = 5
Critical Values				
d.f = 40				
.05 Level	1.46	1.76	1.94	2.07
.01 Level	1.96	2.24	2.41	2.52

V = Ventromedial Hypothalamic Group n = 7

S = Septal Group n = 8

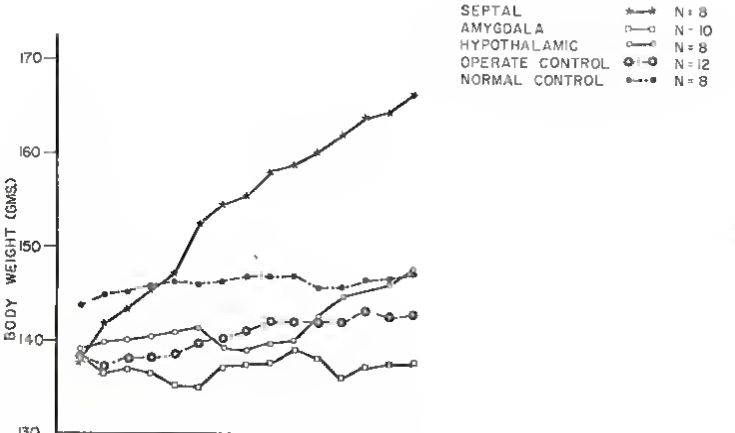
A = Amygdala Group n = 10

OC= Oparata Control Group n = 12

NC= Normal Control Group n = 8

of weight gained or lost from the last preoperative day to the 15th postoperative day produced a significant difference ($H = 33.7$, $P < .001$). The weight change scores are presented in Appendix E. A Kruskal-Wallis test on the last preoperative day on the body weights of the animals failed to show a significant difference between the groups. As with the hoarding scores the weight changes for the three operated controls were pooled after a Kruskal-Wallis test had failed to reveal significant variation between them ($H = 0.8$, $P < .104$) The means for the five groups are shown in Table 7. A Newman-Keuls test revealed that the septals showed a significant increase at the .01 level in the amount of weight gained from all other groups The amygdales showed a decrease at the .05 level of significance from the hypothalamic operates and both controls. Inspection of Figure 3 indicates that the amygdalas do show a somewhat lower body weight level over the postoperative period. The loss of weight found in these animals is reflected in somewhat lower postoperative food intake means as shown in Table 7.

The pre-and postoperative mean water intake scores are presented for each animal in Appendix F. Kruskal-Wallis tests on both preoperative scores and between postoperative scores failed to yield any significant differences Examination of the pre and postoperative water intake means does not reveal any significant trends in water intake



Daily Mean Postoperative Weight Change

Table 7

Mean Weight In Grams Gained or Lost From Last Preoperative Day to Fifteenth Postoperative Day

	Weight Change
Amygdalas n = 10	- 12.3
Septals n = 9	+ 23.2
Ventromedial Hypothalamics n = 7	+ 4.0
Operate Controls n = 12	+ .54
Normal Controls n = 8	+ .25

Table 8

Newman-Kauls Test of Postoperative Mean Weight Changes (Gms.)

		A	NC	OC	V	S
	Means	-12.3	+0.25	+0.54	+4.0	+23.2
A	-12.3	-	12.55*	12.84*	16.3*	35.5**
NC	+ 0.25		-	0.29	3.75	22.95
OC	+ 0.54			-	3.46	22.66
V	+ 4.0				-	19.2
S	+23.2					-

* $P < .05$
** $P < .01$

	r = 2	r = 3	r = 4	r = 5
Critical Values				
d.f. = 40				
.05 Level	10.01	12.04	13.26	14.14
.01 Level	13.37	15.29	16.45	17.25

V = Ventromedial Hypothalamic Group n = 7
S = Septal Group n = 8
A = Amygdala Group n = 10
OC= Operate Control Group n = 12
NC= Normal Control Group n = 8

Nest Ratings

Seventeen hamsters were independently rated on nest building for four preoperative sessions. A Spearman Rank Correlation Coefficient was computed between the scores assigned to each animal by the observers. A coefficient of .92 was obtained between the two observers.

The pre- and postoperative nest rating means for each animal are presented in Appendix G. Ratings are not available for group H-I animals preoperatively. A Kruskal-Wallis test between the septal, amygdala, AC, SC, MC animals did not reveal any significant variation preoperatively. Postoperative ratings were obtained for all animals. A Kruskal-Wallis test of the postoperative mean ratings of all animals produced a significant H value, ($H = 17.7$, $P < .01$). Inspection of Appendix F seems to indicate that only the septal group shows a significant decrease in postoperative nest construction. A Kruskal-Wallis test was run using the postoperative means of all groups except the septal animals, no significant variation was found. It seems fair to conclude that the septal group is responsible for the significant variation between groups. The finding that septal lesions disrupt nesting behavior has already been reported (Matalka, 1964).

Summary of Behavioral Results

1. A significant reduction in postoperative hoarding under ad lib feeding was found in both the septal and hypothalamic groups. Although the amygdala group showed a decrease in postoperative hoarding, their postoperative scores did not differ significantly from the control groups'.

2. Food deprivation failed to increase the postoperative hoarding of the septal and hypothalamic groups to control levels. Although the amygdalas do not significantly differ from the normals postoperatively, they do show a reduction in hoarding.

3. The septal group showed a significant increase in postoperative food intake and also showed a significant weight increase. The amygdalas showed a significant drop in weight postoperatively. No significant changes in weight or food intake were observed in the ventromedial hypothalamic group.

4. A significant decrease in nest building activity was observed in the septal group. The postoperative nesting scores of the amygdala and hypothalamic animals did not significantly differ from that of the control groups.

Histological Results

The Knigge and Joseph (1963) Hamster Atlas was used in the examination of sections.

Ventromedial hypothalamic lesions

All of the lesions in this group were largely confined to the medial hypothalamus. There is some variation in the size and locus of the lesion from animal to animal. The hypothalamic areas which appear to be consistently damaged are the ventromedial nuclei, the dorsomedial nuclei, the arcuate nuclei, the premammillary nuclei, and the periventricular striatum. Other structures that received some degree of damage were the anterior hypothalamus, the paraventricular nuclei, the medial mammillary nucleus, and the posterior

hypothalamus. In addition there appeared to be some unilateral invasion of the medial forebrain bundle in two animals.

Two animals, H-III 8 and H-III 18, suffered total ablation of the ventromedial nuclei. The remaining five animals of this group all showed varying degrees of damage to the dorsal, ventral, medial, and lateral portions of these nuclei. Electrode tracts were clearly visible in only two of the four electrode insertion animals. The tracts appeared to traverse the ventromedial nuclei bilaterally.

Appendix H shows representative sections for animals H-III 8 and H-I 16. H-III 8 had one of the largest lesions and H-I 16 had the smallest.

Septal nuclei lesions

The lesions of all five septal hamsters of group H-II are very similar. The lesions almost totally ablated the medial and lateral septal nuclei pre-and post-commisurally. Post-commisural fornix fibers were disrupted in every animal of this group. Structures such as the caudate nucleus, rostral corpus callosum, anterior commisure, and the fimbria received varying degrees of damage. However, damage to these structures was not consistently observed in the animals of this group. The lesions of the three remaining animals clearly differed from those of group H-II. H-III 11 appeared to have suffered only unilateral damage to the septal area. The lesion was largely confined to the lateral septal nucleus, with the medial nucleus, fornix and fimbria receiving some unilateral damage. Both H-I 4 and H-III 4 had similar lesions. Both lesions were relatively small and were confined

to the pre-commisural septal area. Only the dorsal portions of the lateral and medial septal nuclei were damaged in these animals, there was also slight damage to the rostral corpus callosum.

Representative sections for the animal having one of the smallest lesions, H-III 4, and one of the largest, H-II 7, are shown in Appendix H.

Amygdaloid lesions

Some diversity in the locus and size of the lesion was noted in the amygdaloid group. All of the animals of group H-II and animal H-III 13 appeared to have similar lesions. The damage seemed to be confined to the basolateral complex with the lateralis and centralis nuclei being the most consistently damaged. The stria terminalis, and ventromedial tip of the external capsule also appear to be consistently damaged. The medialis, pars lateral-pars medialis, anterior amygdaloid area, claustrum, internal capsule, pyriform cortex, and optic tract showed some degree of damage. These structures do not appear to be consistently or severely injured in these animals.

Animals H-II 1, H-III 16, H-III 20 had lesions smaller and more ventrally placed than the above animals. The pyriform cortex, and nucleus corticalis, and nucleus medialis were consistently damaged. The claustrum and external capsule appear to show some slight incidental damage.

Representative sections are shown in Appendix H for animals H-II 8 and H-III 16. H-II 8 and H-III 16 had, respectively, one of the largest and one of the smallest lesions of the amygdaloid group.

DISCUSSION

Introduction

The septal, amygdaloid, and ventromedial hypothalamic areas have all been implicated in ingestive behavior to some degree. Little effort has been made to examine the roles of these areas on an unlearned food-motivated behavior such as hoarding. The purpose of this study was to examine the relationship of these areas to feeding and hoarding behavior in the hamster.

Examination of the three lesion groups on the behavioral variables studied indicates that no two groups showed a similar pattern of postoperative changes. The septal group showed severe deficits in hoarding and nesting, while food intake and body weight increased. The ventromedial hypothalamic lesioned Ss showed a significant reduction in hoarding behavior, but body weight, food intake and nesting appeared to be unaffected. The amygdale group showed decreases in body weight and hoarding though only the effect on body weight was significant. Nesting and food intake were not significantly changed. The operated control groups were not significantly different from normal controls on any of the behavioral measures.

Ventromedial Hypothalamic Lesions

Food intake and body weight

The failure to find a postoperative increase in food intake and body weight in the hypothalamic damaged hamster was expected. Skinlety (1966) did not find a consistent relationship between ventromedial hypothalamic damage, postoperative hyperphagia, and obesity. Most researchers, however, have had little difficulty in producing hypothalamic hyperphagia. Obesity in the rat following ventromedial hypothalamic injury was initially reported by Hetherington and Ranson (1940), later authors reported hypothalamic hyperphagia in the cat, (Wheatley, 1944), the dog (Heinbecker and White, 1944), the mouse (Mayer, French and Zighera and Barnett, 1955), the monkey (Hamilton and Brobeck, 1964), and the ground squirrel (Satinoff, 1967).

Although the hypothalamic lesioned hamsters did not show a significant increase in body weight, examination of Appendix E indicates that at least two animals, H-111 8 and H-111 18, show substantial weight increases postoperatively. These two animals also appear to have suffered the most complete ablation of the ventromedial nuclei. Inspection of Appendix E, however, does not indicate that these animals became hyperphagic postoperatively. Hamster H-111 8 does show a postoperative increase in food intake, but it is certainly not a doubling or a tripling of food intake as was reported by Brobeck, Tepperman and Long (1943) following ventromedial hypothalamic lesions.

The postoperative deprivation schedule reduced the body weights of all animals; there is no indication that H-111 8 or H-111 18 showed increased rate of weight gain after being removed from the deprivation schedule. Hoebel and Teitelbaum (1966) have shown that rats made obese by insulin injections tend to show little hyperphagia and weight gain following ventromedial hypothalamic lesions. It might be suggested that the hamsters were already too obese and might not have been expected to show hyperphagia and obesity after hypothalamic damage. H-111 8 and H-111 18 were two of the lightest animals of this group, however, 130 and 121 grams, respectively. On the basis of initial weight, they should have been the most likely to show hyperphagia and concomitant obesity.

There would appear to be two general explanations for the lack of postoperative hyperphagia in the hypothalamic lesioned hamster. First, there is the possibility that a difference exists between the hamster and other species as to the role of the ventromedial hypothalamus and feeding. Second, the hypothalamic lesions did not do sufficient damage to the ventromedial nuclei of the hamsters to produce hyperphagia. There was some variability in the extent to which the ventromedial nuclei was damaged in the animals of this group. The destruction of the ventromedial nuclei seen in H-111 8 and H-111 18, however, should have been more than sufficient to produce hyperphagia in these two animals. Only the production of ventromedial lesions in a larger number of hamsters will resolve the question.

Although Stevenson (1949) has reported hypodipsia in the ventromedial hypothalamic rat, no significant changes were observed in the postoperative water intake of the hypothalamic lesioned hamsters in this study.

Hoarding and nesting behavior

The reduction in hoarding observed in the hypothalamic lesioned hamsters might be explained by the general decrease in locomotor activity found after ventromedial hypothalamic lesions in the rat (Hetherington and Ranson, 1942, Teitelbaum, 1957; Kennedy, 1961; Kennedy and Mitra, 1963). A postoperative drop in activity appears to occur if the medial hypothalamus is lesioned between the preoptic and premammillary regions. The hypoactivity is independent of other effects of the lesion (Gladfelter and Brobeck, 1962, Kennedy and Mitra, 1963).

Although no specific data were collected on the hypothalamic lesioned hamsters of this study, two Ss did show a reduction of activity in the postoperative hoarding trials. Close observation of hamsters H-III 8 and H-III 18 indicated that postoperatively these animals tended to show a severe reduction in the number of entrances to the runway and approaches to the food basket. The animals spent most of their time in grooming, eating, and sleeping. This contrasts with the behavior of hippocampal lesioned hamsters, which also show a significant decrease in hoarding, but maintain a relatively high level of activity.

Postoperative food deprivation had only a slight effect on hoarding; only one animal H-111 18 showed an increase to pre-operative levels. Teitelbaum (1957) showed that the ventromedial lesioned rat compared to normals did not react to food deprivation with an increase in activity. It may well be that the hypothalamic hamsters do not react to food deprivation with an increase in activity even though they did not show hyperphegia postoperatively. It is clear that food deprivation did not produce the increase in hoarding which was seen preoperatively.

Although there is no direct evidence relating temperature to hoarding in the hamster, McCleary and Morgan (1946) found an inverse relationship between temperature and hoarding in the rat. It might be suggested that the deficit in hoarding was related to incidental damage to thermoregulatory structures in the hypothalamus. Kinder (1927) reported a relationship between nest construction and temperature in the rat. The fact that the hypothalamic hamster of this study did not show a significant postoperative change in nest building would argue against damage to hypothalamic thermoregulatory areas. In addition, rectal temperatures obtained from two hamsters with anterior hypothalamic lesions (Matalka and Ellis, unpublished) did not appear to differ from temperatures obtained from normal hamsters. It appears unlikely that disruption of hypothalamic thermoregulatory systems is responsible for the hoarding deficit of the hypothalamic animals.

Septal Lesions

Food intake and body weight

Comparison of the histological results and the weight gains shown in Appendix E indicated that the animals showing a gain of 30 or more grams over the fifteen day postoperative period had large lesions which disrupted post-commissural fornix fibers. The exception to this is hamster H-11 1, which had the largest lesion of this group. A slight postoperative decrease in food intake was observed in this animal. Possibly incidental damage to the thalamus or caudate nucleus might have interfered with ingestive behavior and somewhat retarded weight gain. Increases in food intake have been reported in the rat following fornix lesions (Ehrlich, 1963) and hippocampal lesions (Kimble and Coover, 1966). Postoperative increases in food intake did not lead to obesity in either the fornix or the hippocampal damaged rats.

There is a significant increase in the postoperative food intake of the septals compared to the lesion and control groups. However, the septals show only a 20% increase in postoperative food intake. There is a relatively slight increase compared to that seen following ventromedial hypothalamic lesions in those studies where hypothalamic hyperphagia has been reported. It is possible that other factors, such as a decrease in activity, may contribute to the development of obesity in the septal hamster. Bougles and Raphelsen (1966) reported an increase in exploratory activity in the septal rat, but a decrease in locomotor activity. Although Matalka (1964) did not find a

significant change in exploratory activity in the septal lesioned hamster it may be that septal lesions produce a similar decrease on locomotor activity in the hamster.

Harvey and Hunt (1965), Carey (1967), Pizzi and Corens (1967) have reported postoperative increase in the water intake of the septal rat. No significant changes were found in the water intake of the septal lesioned hamster. The hamster, being a desert dwelling animal, may well have evolved somewhat different mechanisms of water intake and would not be effected by a septal lesion in the same fashion as the rat. It is also possible that the authors reporting increases in water intake damaged other neural structures in addition to the septal area. The lesion which Carey (1967) reported as having produced an increase in water intake is quite ventrally placed for a septal lesion. It would seem likely that this type of lesion would damage the preoptic region. A second type of lesion appeared to be more closely confined to the septal region. No increase in water intake appeared in rats with this type of lesion. The septal lesions that Pizzi and Coren (1967) and Harvey and Hunt (1965) describe as having produced increased ingestion of water appear to damage more ventrally placed structures such as the preoptic area. The lesion seen in the septal hamsters of this study more closely resembled those which failed to increase water intake in Carey's study.

Hoarding and nesting behavior

The septal animals suffered severe postoperative reductions in hoarding. Hoarding behavior was totally abolished in those animals

that received large lesions damaging the post-commissural fornix. Some sparing of the behavior was observed in hamsters H-III 11, H-III 4, and H-I 4. These animals suffered little or no damage to the post-commissural fornix. The septals like the hypothalamic group showed an apparent decrease of activity in the hoarding situation.

Observation of hamsters H-III 4 and H-III 11 indicated that both animals showed a postoperative decrement in the number of runway entrances and basket approaches during the hoarding trials. This again would seem to be compatible with the decrease in locomotor activity reported by Douglas and Raphelson (1966).

As mentioned above, hippocampal and cingulate lesioned hamsters show a postoperative decrease in amount of pellets hoarded, but show a considerable amount of activity during the hoarding trials. The nature of the deficit in cingulate and hippocampal animals appears to be more a disruption of sequential behavior where the septals may be showing a general lower level of activity.

Food deprivation did not restore the hoarding behavior of the septals postoperatively. In fact, it appeared to have virtually no effect at all. Most of the septal group had gained considerable amounts of weight by the time the deprivation trials were begun. It is possible that the septals would have to be reduced to their preoperative weight levels in order for hoarding to be restored. One difficulty in attempting to restore hoarding behavior by food deprivation is the fact that the septals can spend the hoarding trial

eating rather than hoarding. A more effective way of restoring hoarding in these animals might be to reduce environmental temperature.

The disruption of nesting behavior exhibited by septal hamsters (Matalka, 1964) has been confirmed and extended by this study. Examination of Appendix G indicates that, as with hoarding, the animals showing the greatest deficits are also those with lesions severing the post-commissural fornix. In fact, it is questionable that hamsters H-III 4 and H-III 11 even show a deficit in nesting. The apparent involvement of the post-commissural fornix in nesting suggests that the hippocampal ablations in both the rat (Kim, 1960) and hamster (Bunnell and Goodman, unpublished), Scopalamine, a drug believed to reproduce certain effects of hippocampal ablation, (Douglas and Isaacson, 1966) also produces a deficit in the nesting behavior of the hamster (Burkett, Matalka and Ellis, unpublished).

There remains the possibility that the deficit in nesting is due to changes in thermoregulation produced by septal damage. Stuart, Kawamura, Hemingway, and Price (1962) were unable to find any changes in heat loss or shivering following septal lesions in the cat. In general, there appears to be little evidence to implicate the septal area in temperature regulation.

Amygdala Lesions

Food intake and body weight

The reduction in food intake and significant decrease in body weight seen in the amygdaloid lesioned animals is similar to the

affects of amygdaloid lesions in the rat. Anand and Brobeck (1952) reported transient weight losses and mild aphagia in some of their amygdaloid lesioned rats. Yamada and Greer (1960) reported severe aphagia and weight loss following amygdaloid lesions in the rat. Kling and Schwartz (1961) reported persistent aphagia after bilateral amygdaloid damage in infant rats. Kolkegami, Fuse, Hiroki, Kazami, and Kageyama (1958) found similar effects following amygdalectomy in young rats and puppies. Grossman and Grossman (1964) reported an increase in food intake, but not an increase in body weight following posterior amygdala lesions. With the exception of Grossman and Grossman, however, most researchers reporting hyperphagia after amygdaloid lesions have used the cat (Morgane and Kosman, 1959, Wood, 1958, Kolkegami, Fuse, Hiroki, Kazami, and Kageyama, 1958). It is possible that a real species difference exists between the rodent and cat with respect to the function of the amygdala in ingestive behavior.

The effects of amygdaloid lesions on the body weight and food intake of the hamster does not appear to be as severe as that observed by Yamada and Greer (1960), Kling and Schwartz (1961), and Kolkegami et al. (1958). The body weights of every amygdaloid animal showed a postoperative decrease. Large drops in weight were observed in hamsters H-11 14 and H-11 17. Both of these animals, particularly H-11 14, showed substantial postoperative decreases in food intake. Both of these animals suffered unilateral damage to the internal capsule. It is interesting to note that Gold (1967) has reported persistent

aphagia following unilateral lesions in the internal capsule. The postoperative deprivation schedule again interferes with the weight gains of these animals. Though most of the animals, with the exception of H-II 14 and H-II 17, showed some upward trend in body weight only H-III 1 ever exceeded the weight recorded immediately prior to operation.

Hoarding and nesting behavior

The amygdaloid group suffered a significant drop from their preoperative hoarding level but did not differ from the normal controls postoperatively. The reduction in hoarding observed in the amygdaloid group might be due to a decrease in activity similar to that reported in rats after amygdaloid damage (Anand and Brobeck, 1952). Observation of the number of runway entrances and basket approaches for animals H-III 1, H-III 13, H-III 16, and H-III 20 indicated that though these animals showed a slight drop in postoperative activity in the hoarding situation, the decrement in activity is by no means as severe as that seen in the hypothalamics and septals. Unfortunately, animals H-III 1, H-III 16, and H-III 20 had somewhat smaller and more ventrally placed lesions than the lesions of the remaining animals of the amygdaloid group, so they may not be representative of the entire group. In general, the amygdaloid animals maintained a relatively high level of activity compared to the other lesion groups. It is more likely that the drop in hoarding and activity is secondary to the decreases in body weight exhibited by several of the animals of this group. A

further drop in hoarding was exhibited by hamsters H-11 3, H-11 6, H-11 14 and H-11 17, nor did H-11 8 and H-11 19 show substantial increases. This is what one would expect to find if physically weak animals were further weakened by deprivation.

Bunnell, Shalloway, and Sudatz (1965) reported an increase in hoarding in amygdaloid lesioned hamstars. The fact that the amygdaloid hamstars in this study had a longer distance to travel between the food pellets and their home cages added to the decreasa in body weights observed and may explain the contradiction between these two studies.

Anand and Brobeck (1952) reported temporary hypothermia following amygdaloid lesions in the rat. The fact that postoperative nest building remained unchanged provides indirect evidence for normal temperature regulation in the amygdaloid lesioned hamstar.

Theoretical Discussion

Glickman and Schiff (1967) have developed a general theory of reinforcement which might be applicable to the results of this study. In essence their theory proposes that reinforcement has developed as a biological mechanism to insure species typical responses to appropriate stimuli. Glickman and Schiff have followed Schneirla's (1959) classification of behavior into approach or withdrawal patterns. Glickman and Schiff consider feeding and drinking, maternal care, sexual activity, and investigatory activity to be examples of approach behaviors. Withdrawal patterns constitute avoidance or escape from

various types of noxious stimuli. Although Glickman and Schiff do not specifically discuss hoarding there is little doubt that this behavior would fall under the approach category of behavior.

Glickman and Schiff have identified the approach behaviors with the lateral hypothalamus and medial forebrain bundle. High rates of self-stimulation or feeding can be obtained from stimulation of lateral hypothalamus (Margules and Olds, 1962). Roberts and Carey (1965) have elicited gnawing and nest building responses from stimulation of this area. Herberg and Blundell (1967) have produced hoarding in the rat by lateral hypothalamic stimulation. Olds (1958) has reported high rates of self-stimulation from electrode implants in the septal area. Robinson (1964) elicited components of ingestive behavior upon stimulation of the septal region in the monkey. These studies, plus the anatomical connections between the septum and medial forebrain bundle (Bullery, 1959, Nauta, 1956, Nauta, 1959, Valenstein and Nauta, 1959), would tend to indicate that the septal area should be included in the approach system of Glickman and Schiff.

The deficits observed in the hoarding and nesting behavior of the septal lesioned hamster might then be explained in terms of a disruption of the approach system postulated by Glickman and Schiff. Septal lesions may interfere with impulses from forebrain structures which act to initiate and guide behaviors such as hoarding and nest-building. However, the septal hamster shows an increase in food intake. One would expect on the basis of Glickman and Schiff's theory to find a disruption in feeding as well as hoarding and nesting. Glickman

and Schiff have not distinguished between consumatory and appetitive behaviors in developing their theory. In fact, they have ascribed reinforcing properties to the approach-withdrawal behavior sequences rather than restricting reinforcement to consumatory responses such as feeding and drinking. Miller (1955) has suggested that brain lesions can increase eating but decrease food motivation. The results of the septal group generally agree with Miller's hypothesis, feeding was increased and a food motivated behavior, hoarding, was decreased. It would appear that Glickman and Schiff's theory should be modified to draw some distinction between eating and a food motivated response.

Although Glickman and Schiff discuss the possible function of the amygdala with respect to approach-withdrawal behavioral sequences, they are not particularly specific about its relationship to these behaviors. They do suggest that the amygdala acts as some sort of sensory filter which, "mediates the affects of biologically salient stimuli." This concept would not appear to be compatible with the results of this study. The amygdaloid lesioned hamsters were certainly capable of responding to the appropriate sensory stimuli for hoarding which are likely visual and olfactory in nature. Douglas and Pribram (1966) implicate the amygdala in a reinforcement-registration process. The gist of Pribram and Douglas' theory would seem to be that the amygdala is important in the initial registration of some experience closely followed by biologically significant, i.e. "reinforcing" event. They term the residual impact "impellance" which

apparently alters the manner in which the experience will be perceived by the organism. The amygdala would appear to be involved in the increase of impellance but not necessarily in its storage. It would appear then that behavior acquired prior to amygdaloid damage would not be expected to be seriously disrupted postoperatively. In general, this concept of amygdala function would better fit the results of the amygdaloid lesioned hamsters on hoarding than the Glickman and Schiff's concept.

Both approach and avoidance behaviors have been elicited by medial hypothalamic stimulation (Bower and Miller, 1958). In addition, negative and positive effects have been reported for self-stimulation in the medial hypothalamus (Poschel, 1959). The apparent mixing of approach-withdrawal responses in the medial hypothalamus makes it difficult to predict the effects of lesions in this area. The reduction in hoarding found in the hypothalamic lesioned hamsters would suggest that damage to this region disrupts approach type behaviors. The fact that nesting and feeding were unaffected following medial hypothalamic damage contradicts this suggestion. The hypoactivity reported to accompany medial hypothalamic damage provides a better explanation for the hoarding deficit seen in the hypothalamic hamster. One would expect that behaviors requiring considerable activity, such as hoarding, would be seriously affected by lesions in this area. Behaviors requiring less physical activity like nest building would likely remain unaffected.

Comparison of Lesion Effects

Lesions in the medial hypothalamus did not produce a pattern of changes similar to that seen after septal or amygdaloid lesions. This would argue against a functional relationship between the medial hypothalamus and either of these two limbic areas. Both septal and hypothalamic lesions produced similar decreases in hoarding and both types of lesions have been reported to reduce activity which may cause the deficit. The disruption in nesting and increased food intake seen only in the septals argues for a more general effect of septal lesions on appetitive and consumatory responses, rather than activity alone. Except for the fact that nesting was unaffected by amygdaloid and hypothalamic lesions, the two groups were not similar. It was hypothesized that the septal and amygdaloid areas might play a reciprocal role in hoarding. The amygdaloid lesions did not produce the increase in hoarding that would be expected under such a hypothesis. However, the amygdaloids may not have been physically capable of demonstrating an increase in hoarding due to the weight loss this group suffered postoparatively.

The increase in feeding and body weight in the septal group and general decrease in these variables seen in the amygdala group indicate a possible reciprocal relationship between these areas on the feeding behavior of the hamster. Since medial hypothalamic lesions did not significantly affect eating, these areas may exert their influence on lateral hypothalamic or midbrain regions.

Hoarding and feeding were independently affected by the lesions. Septal lesions increased eating but decreased hoarding. The hypothalamic group did not show a significant change in food intake, but also exhibited a clear decrease in hoarding. The amygdaloids hoarded more than the septal or hypothalamics but showed a reduction in food intake and a significant decrease in body weight. This apparent dissociation of feeding and hoarding suggests that these behaviors are subserved by separable neural systems.

Conclusions

Some conclusions can be suggested on the basis of the results of this study.

A clear decrease was observed in the postoperative hoarding of the hypothalamic hamsters. These animals appeared to show a decrease of activity in the hoarding situation, which is in agreement with reports of decreased locomotor activity following medial hypothalamic damage. The lack of a postoperative deficit in nesting indicates that species' typical responses in general are not disrupted. This suggests that the reduction in hoarding is likely the result of a decrease in activity commonly found after medial hypothalamic damage.

The fact that the hypothalamic lesions failed to produce hyperphagia is surprising. The degree of injury suffered by the ventromedial nuclei varied somewhat from animal to animal. Two animals showed almost total destruction of the ventromedial nuclei and should have demonstrated hyperphagia. A difference may exist between the

ventromadial hypothalamus of the hamster and other species with respect to food intake.

The results of this study confirm earlier reports of obesity in the septal lesioned hamster. Food intake measures indicated that the septal hamster shows evidence of hyperphagia. Other factors may also be important in the development of obesity. A postoperative reduction in activity may also contribute to the development of obesity. A reduction in activity may well be responsible for the postoperative hoarding deficit seen in these animals. The deficit observed in nesting makes it more probable that septal lesions produce a disruption of approach-type behaviors as described by Glickman and Schiff (1967). The greatest increases in food intake and weight, and conversely the greatest decreases in nesting and hoarding are associated with post-commissural septal damage.

Amygdaloid lesions in the hamster seem to produce a decrease in body weight similar to that seen in the rat. The results of the hoarding tests did not confirm the Gunnell, Shalloway, Sodetz (1965) finding that amygdaloid lesions increased hoarding in the hamster. In fact, a decrease in hoarding was observed in this group. The reduction in weight may have produced inanition which rendered these animals incapable of exhibiting a high level of hoarding. The additional reduction in hoarding seen in most of these animals during the postoperative deprivation trials tends to confirm this view.

The results did not confirm the hypothesis of a reciprocal relationship between the septal and amygdaloid areas with respect to hoarding. This statement must be qualified in view of the weight losses seen in the amygdaloids. The increase in food intake and body weight seen after septal lesions and decrease in body weight and food intake after amygdaloid damage, suggest a reciprocal relationship between these areas on feeding behavior.

No direct functional relationships appear to exist between septum and amygdala and the medial hypothalamus for the behaviors studied.

SUMMARY

This study investigated the effects of septal, amygdaloid, and hypothalamic lesions on the feeding and hoarding behavior of the Syrian Golden hamster. The results of the previous studies indicated that the limbic and hypothalamic lesions might have the following effects: (1) septal lesions would decrease hoarding and increase food intake; (2) amygdaloid lesions would increase hoarding and decrease food intake, (3) ventromedial hypothalamic lesions would decrease hoarding and increase food intake. It was further hypothesized that the septal and amygdaloid areas might play reciprocal inhibition roles in the regulation of hoarding.

Eight septals, ten amygdalas, seven ventromedial hypothalamics, eight normals, and twelve operated controls were tested pre and postoperatively on hoarding. Daily food and water intakes and nest ratings were obtained for all animals.

Septal forebrain lesions produced the predicted changes. Hoarding was significantly decreased and postoperative food intake increased. The septals became obese. Amygdala lesions failed to increase hoarding. A significant reduction in body weight was noted in the amygdalas. Postoperatively the food intake of the amygdalas was reduced, although the decrease was not statistically significant. The ventromedial hypothalamics showed a significant decrease in hoarding, but failed to exhibit a significant increase in food intake.

Nest building was significantly reduced in the septals, but was uneffected by hypothalamic or amygdaloid lesions. No significant changes were seen in the water intakes of the lesion or control animals.

The deficits in hoarding and nesting suggest that septal lesions produce a disruption of "appetitive" behaviors, while increasing the consumetory response of eating. Similar effects have been reported following hypothalamic lesions in the rat. The body weight and food intake reductions seen after amygdaloid lesions are similar to findings in the rat. It was suggested that the failure of the amygdaloids to show increased hoarding was secondary to the inanition produced by weight loss.

The pronounced deficit in hoarding seen in the hypothalamic group was related to the general decrease in activity seen in the rat following medial hypothalamic damage. The failure to find an increase in food intake following ventromedial hypothalamic damage indicates that this area may not be involved in the regulation of feeding in the hamster.

The results of this study failed to confirm the hypothesis that the septal and amygdaloid areas play reciprocal roles in the regulation of hoarding. The changes in food intake produced by these lesions suggested that these areas might play a reciprocal role with respect to feeding. The results of the study indicate that brain lesions can independently affect feeding and hoarding. The results of this

study failed to show a firm functional relationship between the septal, amygdaloid, and ventromedial hypothalamic areas and the behaviors studied.

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APPENDICES

APPENDIX A

NESTING ACTIVITY SCALE

0 - PAPER SCATTERED

1 - PAPER ACCUMULATION TOWARD REAR OF CAGE

2 - PALLET IN REAR OF CAGE, BUT CONSIDERABLE PAPER STILL SCATTERED

3 - PALLET, BUT RAGGED ABOUT THE EDGES

4 - PALLET, EDGES NEAT, NO PAPER SCATTERED

5 - PALLET, SIDES AGAINST REAR AND SIDE WALL

6 - NEST WITH SIDES ALL AROUND

7 - NEST WITH SIDES AND TOP

APPENDIX B

Total Weight of Pellets Hoarded (Gms.) for Individual Animals Over the Pre- and Postoperative Hoarding Trials under Ad Lib Feeding.

Group	Preop	Postop	Group	Preop	Postop
Amygdala			Septal		
H-II 3	4082.0	2110.0	H-I 4	953.0	301.0
H-II 6	4254.0	2017.0	H-II 1	5009.0	0.0
H-II 8	3014.0	472.0	H-II 2	4285.0	0.0
H-II 14	3263.0	558.0	H-II 7	5103.0	5.0
H-II 17	4185.0	1023.0	H-II 13	4552.0	0.0
H-II 19	1025.0	716.0	H-II 15	4271.0	16.0
H-III 1	14.0	500.0	H-III 4	953.0	301.0
H-III 13	4420.0	2843.0	H-III 11	2315.0	1227.0
H-III 16	3610.0	3917.0			
Amygdaloid			Septal		
Control			Control		
H-II 9	3696.0	2278.0	H-II 11	6228.0	2428.0
H-II 16	4965.0	3583.0	H-II 20	459.0	812.0
H-III 14	2786.0	2012.0	H-III 3	736.0	6143.0
H-III 17	130.0	112.0	H-III 12	2780.0	3242.0

APPENDIX B (Continued)

Group	Preop	Postop
Hypothalamic		
H-I 2	3261.0	642.0
H-I 3	1231.0	182.0
H-I 7	2893.0	137.0
H-I 14	4410.0	10.0
H-I 16	2840.0	2.0
H-III 8	947.0	293.0
H-III 18	276.0	0.0

Group	Preop	Postop
Normal		
H-I 5	1697.0	2303.0
H-I 15	2672.0	996.0
H-II 5	3263.0	3055.0
H-II 18	960.0	543.0
H-III 2	6132.0	6352.0
H-III 6	1431.0	1267.0
H-III 9	1904.0	302.0
H-III 19	1398.0	2754.0

Hypothalamic		
Control		
H-I 6	5432.0	534.0
H-I 12	1813.0	19.0
H-III 5	2528.0	767.0
H-III 7	2830.0	2437.0

APPENDIX C

Total Weight of Pellets Hoarded (Gms) for Individual Animals over the Pre- and Postoperative Hoarding Trials under Food Deprivation

Group	Preop	Postop	Group	Preop	Postop
Amygdala			Septal		
H-II 3	2892 0	334 0	H-I 4	548 0	88 0
H-II 6	2221 0	490 0	H-II 1	1914.0	0 0
H-II 8	1641 0	346 0	H-II 2	2103 0	0 0
H-II 14	2305 0	174 0	H-II 7	2185 0	0 0
H-II 17	2155.0	360 0	H-II 13	2221 0	0 0
H-II 19	999 0	384 0	H-II 15	2911 0	15 0
H-III 1	1412 0	715 0	H-III 4	2405 0	475 0
H-III 13	2279 0	2519 0	H-III 11	3084 0	1284 0
H-III 16	3051 0	2561 0			
H-III 20	764 0	273 0			
Amygdala			Septal		
Control			Control		
H-II 9	6610 0	857 0	H-II 11	2457 0	2057 0
H-II 16	2801 0	2439 0	H-II 20	1549 0	2474 0
H-III 14	2685 0	1713 0	H-III 3	1910 0	2534 0
H-III 17	430.0	1354.0	H-III 12	2914 0	2769 0

APPENDIX C

Group	Preop.	Postop.
Hypothalamic		
H-I 2	2977.0	466.0
H-I 3	1103.0	172.0
H-I 7	1503.0	224.0
H-I 14	2857.0	0.0
H-I 16	2345.0	25.0
H-III 8	2434.0	364.0
H-III 18	394.0	1099.0
Hypothalamic		
Control		
H-I 6	2951.0	682.0
H-I 12	95.0	447.0
H-III 5	1970.0	1239.0
H-III 7	2282.0	2604.0

Group	Preop	Postop
Normal		
H-I 5	973.0	2160.0
H-I 15	2056.0	725.0
H-II 5	2912.0	1715.0
H-II 18	1708.0	712.0
H-III 2	3040.0	3137.0
H-III 6	1821.0	564.0
H-III 9	2336.0	564.0
H-III 19	291.0	1355.0

APPENDIX O

Pre- and Postoperative Mean Food Intake [illegible]
for Individual Animals

Group	Preop	Postop	Group	Preop	Postop.
Amygdala			Septal		
H-11 3	9 8	7.2	H-1 4	11.5	9 0
H-11 6	11.6	7.8	H-11 1	9 2	14 1
H-11 8	12.3	6 6	H-11 2	7 6	13.5
H-11 14	10 5	3 3	H-11 7	9.5	9.7
H-11 17	12 0	6 7	H-11 13	10.0	13 5
H-11 19	12 4	7 7	H-11 15	10.7	12 1
H-111 1	8 3	7 1	H-11 14	6.4	8 5
H-111 13	7.7	7.2	H-111 11	8.9	9 4
H-111 16	8.1	7 0			
H-111 20	9 3	8.4			
Amygdala			Septal		
Control			Control		
H-11 9	12.1	7.2	H-11 11	9.0	7 2
H-11 16	11 6	8 9	H-11 20	12.1	8.1
H-111 14	7 2	6 8	H-111 3	7 5	6 9
H-111 17	8 3	7.9	H-111 12	8.3	7.0

APPENDIX D (Continued)

Group	Preop	Postop
Hypothalamic		
H-I 2	11.0	8.2
H-I 3	11.6	6.5
H-I 7	8.0	6.0
H-I 14	9.5	8.4
H-I 16	11.8	10.3
H-III 8	7.9	11.1
H-III 18	8.0	8.1
Hypothalamic		
Control		
H-I 6	11.2	10.1
H-I 12	6.7	4.5
H-III 5	8.4	7.6
H-III 7	8.8	9.7

Group	Preop	Postop
Normal		
N-I 5	10.2	10.0
H-I 15	11.9	9.5
H-II 5	10.5	8.7
H-II 18	12.1	9.3
H-III 2	8.3	8.7
H-III 6	8.6	7.9
H-III 9	7.3	7.6
H-III 19	7.9	8.5

APPENDIX E

Weight Change (Gms.) for Individual Animals

Over a Fifteen Day Postoperative Period

Group	Weight Change	Group	Weight Change
Amygdala		Septal	
H-II 3	- 15.5	H-I 4	- 2.5
H-II 6	- 4.0	H-II 1	+ 33.0
H-II 8	- 19.5	H-II 2	+ 41.5
H-II 14	- 32.5	H-II 7	+ 11.0
H-II 17	- 32.5	H-II 13	+ 38.5
H-II 19	- 8.0	H-II 15	+ 44.5
H-III 1	- 2.0	H-I 4	- 2.5
H-III 13	- 4.0	H-III 4	+ 3.5
H-III 16	- 2.5	H-III 11	+ 10.0
H-III 20	- 2.5		
Amygdala		Septal	
Control		Control	
H-II 9	- 3.0	H-II 11	- 1.0
H-II 16	- 6.0	H-II 20	+ 2.0
H-III 14	- 2.5	H-III 3	+ 1.5
H-III 17	+ 6.5	H-III 12	+ 6.0

APPENDIX E (Continued)

Group	Weight Change	Group	Weight Change
Hypothalamic		Normal	
H-I 1	- 9.5	H-I 5	- 6.5
H-I 2	-15.5	H-I 15	- 0.5
H-I 7	+ 9.5	H-II 5	- 4.5
H-I 14	+ 9.5	H-II 15	+ 2.5
H-I 16	- 3.0	H-III 2	+ 4.5
H-III 8	+21.0	H-III 6	+ 2.0
H-III 18	+16.0	H-III 9	- 1.0
		H-III 19	+ 5.5
Hypothalamic			
Control			
H-I 6	- 4.0		
H-I 12	-13.5		
H-III 5	+ 8.5		
H-III 7	+12.5		

APPENDIX F

Pre- and Postoperative Mean Water Intake [illegible]

for Individual Animals.

Group	Preop.	Postop.	Group	Preop.	Postop.
Amygdala			Septal		
H-11 3	11.0	13.0	H-1 4	7.4	12.6
H-11 6	16.5	14.2	H-11 1	16.3	14.3
H-11 8	10.0	7.3	H-11 2	4.5	12.1
H-11 14	12.8	9.6	H-11 7	11.0	10.7
H-11 17	14.8	4.4	H-11 13	9.5	14.0
H-11 19	10.8	9.2	H-11 15	26.5	11.8
H-111 1	8.5	8.0	H-111 4	7.5	6.3
H-111 13	16.3	10.0	H-111 11	7.8	8.8
H-111 16	13.0	13.5			
H-111 20	15.3	11.3			
Amygdala			Septal		
Control			Control		
H-11 9	13.5	9.0	H-11 11	11.5	13.7
H-11 16	14.3	13.5	H-11 20	4.0	16.6
H-111 14	16.0	9.0	H-111 3	7.0	5.8
H-111 17	35.0	11.3	H-111 12	28.5	17.3

APPENDIX F (Continued)

Group	Preop.	Postop.
Hypothalamic		
H-I 2	11.2	5.6
H-I 3	11 2	6 8
H-I 7	6 4	7.6
H-I 14	9 6	17 4
H-I 16	7.0	9.8
H-III 8	8 0	12.0
H-III 18	8.1	15 3
Hypothalamic		
Control		
H-I 6	8.4	8 0
H-I 12	7.0	5.8
H-III 5	22.0	7.3
H-III 7	25 0	17.0

Group	Preop	Postop
Normal		
H-I 5	27.2	10.8
H-I 15	11_0	18 8
H-II 5	29.0	7.9
H-II 18	11.5	10.4
H-III 2	13.8	19 8
H-III 6	9.3	5.5
H-III 9	14.3	6.8
H-III 19	8.3	12.3

APPENDIX G

Pre- and Postoperative Mean Nest Ac[illegible]

Individual Animals.

Group	Preop.	Postop.	Group	Preop.	Postop.
Amygdala			Septals		
H-11 3	3.8	5 7	H-1 4	-	2 0
H-11 6	6.0	5 5	H-11 1	4 2	0 1
H-11 8	5.6	6.0	H-11 2	3 6	0 5
H-11 14	4.0	3.4	H-11 7	4.8	0.4
H-11 17	5 2	3 6	H-11 13	5.2	0.4
H-11 19	4.8	5.0	H-11 15	5.2	0.8
H-111 1	6.0	5.9	H-111 4	6.0	5.9
H-111 13	5 4	4.5	H-111 11	5.6	5.7
H-111 16	5.4	5.6			
H-111 20	5.0	5.5			
Amygdala			Septal		
Control			Control		
H-11 9	4.0	5.7	H-11 11	4.8	5 7
H-11 16	5.2	5 8	H-11 20	5.4	5 7
H-111 3	5.8	6.3	H-111 3	5.8	6.3
H-111 12	5.8	5.3	H-111 12	5.8	5 3

APPENDIX G (Continued)

Group	Preop.	Postop
Hypothalamic		
H-I 2	-	6.0
H-I 3	-	5.8
H-I 7	-	5.8
H-I 14	-	5.4
H-I 16	-	4.8
H-III 8	6.0	5.5
H-III 18	5.8	5.5

Group	Preop.	Postop.
Normal		
H-I 5	-	6.0
H-I 15	-	6.0
H-II 5	4.2	5.7
H-II 18	4.6	5.8
H-III 2	5.4	5.8
H-III 6	5.8	5.9
H-III 9	6.0	6.0
H-III 19	5.6	5.2

Hypothalamic		
Control		
H-I 6	-	5.6
H-I 12	-	6.0
H-III 5	5.4	5.8
H-III 7	5.4	5.1

APPENDIX H

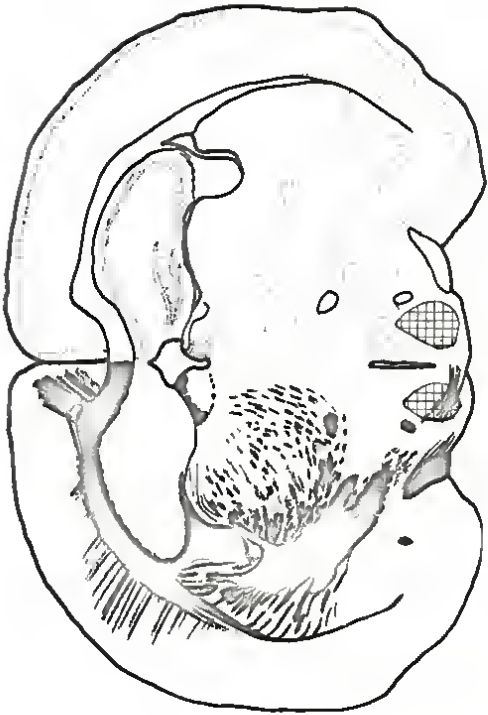
Diagrams of the Hypothalamic, Septal, and Amygdaloid Lesions.

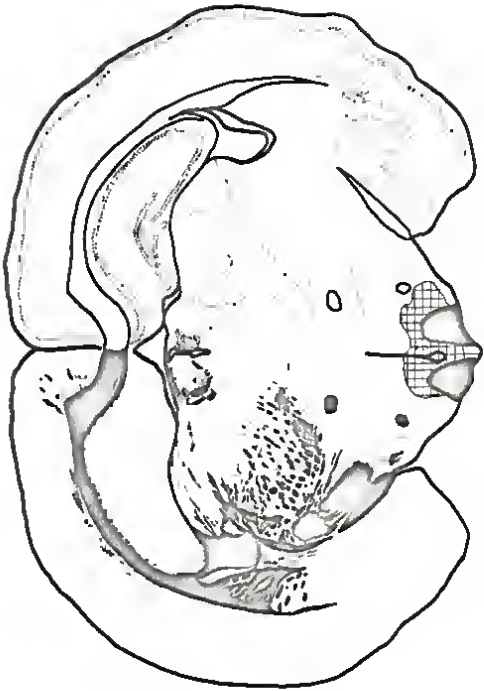
Largest Lesion - 

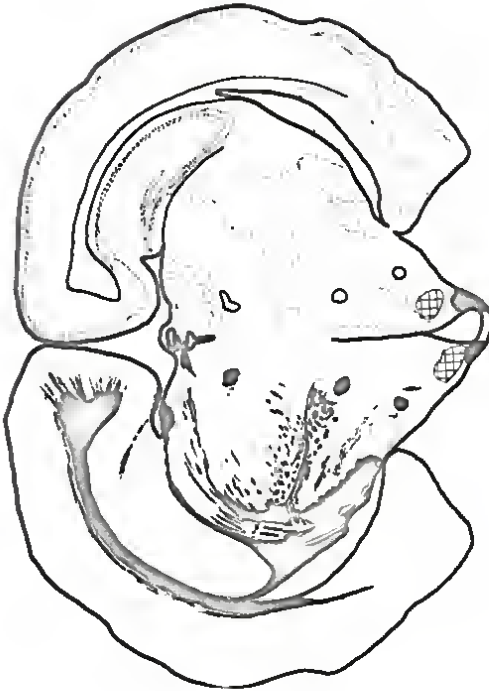
Smallest Lesion - 

Hypothalamic H-111 8

Hypothalamic H-1 16

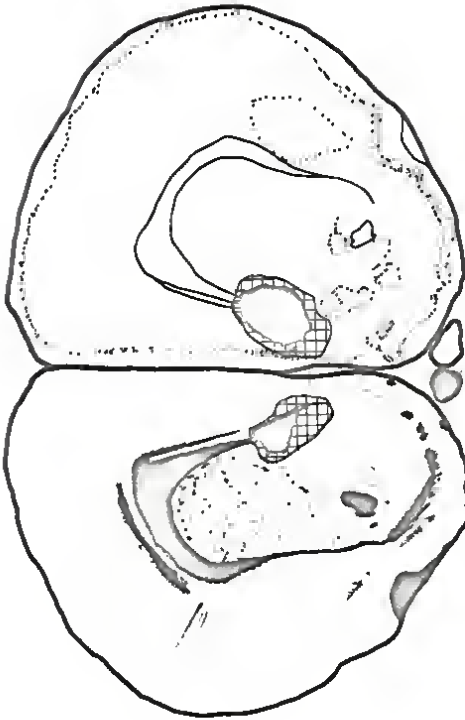


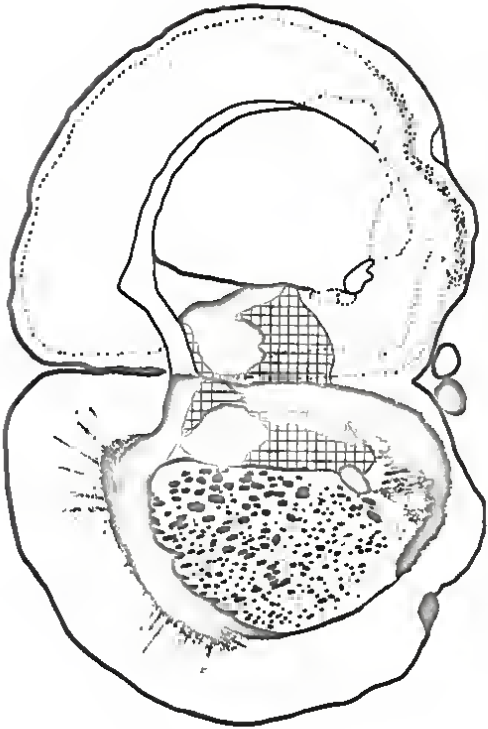


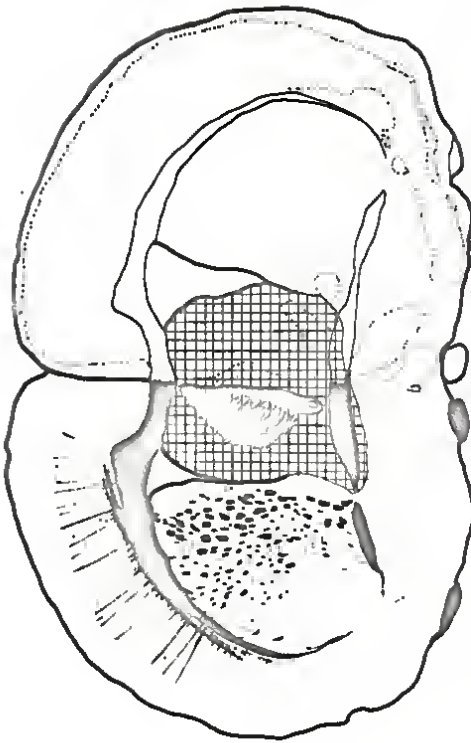


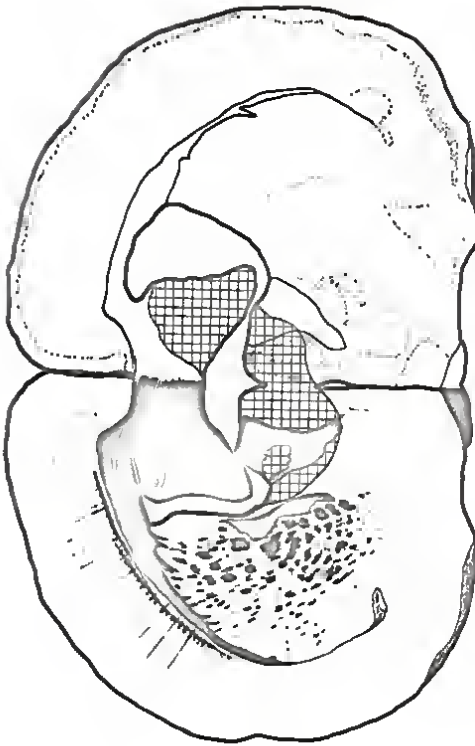
Septal H-II 7

Septal H-III 4



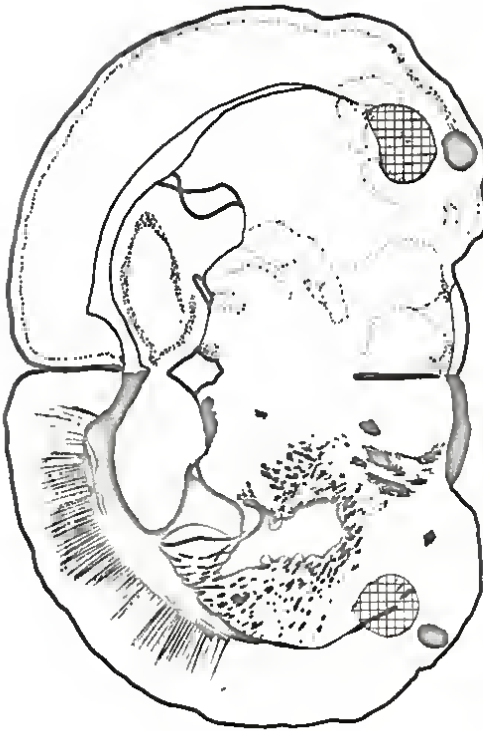


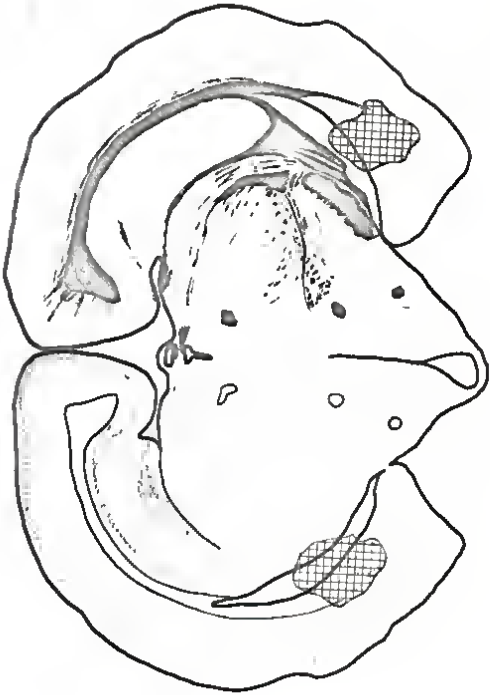




Amygdala H-11 8

Amygdala H-111 16





BIOGRAPHICAL SKETCH

Edward Stephen Matalka was born October 16, 1937, in Binghamton, New York. He attended public schools in Binghamton, graduating from Binghamton North High School in 1955. From 1955 to 1958, Mr. Matalka attended Rensselaer Polytechnic Institute. From 1958 to 1959, he was employed at Link Aviation Company, Hillcrest, New York. Mr. Matalka returned to Rensselaer Polytechnic Institute in 1959 and received a B.S. in June, 1961.

Mr. Matalka entered the Graduate School at the University of Florida September, 1961. From 1961 to 1966, Mr. Matalka was a research assistant in the Department of Psychology. From 1966 to 1967, he was a predoctoral fellow of the Neurobiological Sciences Center of the University of Florida Medical School. Mr. Matalka received a M.S. degree with major in Psychology in December, 1964.

In December, 1965, Mr. Matalka was admitted to candidacy for the Doctor of Philosophy degree. Since that time, he has been engaged in predoctoral research in the area of his specialty, Physiological Psychology.

This dissertation was prepared under the direction of the chairman of the candidate's supervisory committee and has been approved by all members of that committee. It was submitted to the Dean of the College of Arts and Sciences and to the Graduate Council, and was approved as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

August, 1967

Ernest H. Cox
Dean, College of Arts and Sciences

Dean, Graduate School

Supervisory Committee:

Chairman